Final Tier 2 Rule: Air Quality Estimation, Selected Health and Welfare Benefits Methods, and Benefit analysis Results

EPA420-R-99-032

December 1999

Prepared for
Office of Air Quality Planning and Standards

U.S. Environmental Protection Agency

Research Triangle Park, NC

Prepared by
Lisa Akeson
Kenneth Davidson
Leland Deck
Brad Firlie
Emily King
Stephen Lange
Don McCubbin
Ellen Post

Work funded through
Contract No. 68-D-98-001
Work Assignment 2-37
Lisa Conner, Work Assignment Manager
Nancy Riley, Project Officer

DISCLAIMER

This document was developed by Abt Associates Inc. under technical direction from U.S. EPA's Office of Air Quality Planning and Standards. The analysis and conclusions presented in this report are those of the authors and should not be interpreted as necessarily reflecting the official views or policies of the U.S. EPA. The analysis is useful to derive estimates of air quality, costs, benefits, and/or economic impacts. However, the analysis inputs and outputs associated with any emissions source, county, or local area are subject to significant uncertainties and should not be used to predict attainment status, costs, benefits, and/or economic impacts at this level of detail.

ACKNOWLEDGEMENTS

The Work Assignment Manager, Lisa Conner, as well as Bryan Hubbell, Tyler Fox, Scott Mathias, and Norm Possiel of the U.S. Environmental Protection Agency, provided a variety of constructive suggestions, comments, and technical direction at all stages of work on this report.

FINAL TIER 2 RULE: AIR QUALITY ESTIMATION, SELECTED HEALTH AND WELFARE BENEFITS METHODS, AND BENEFIT ANALYSIS RESULTS

TABLE OF CONTENTS

List of Exhibits

1 INTRODUCTION

In July 1998, the U.S. Environmental Protection Agency (EPA) submitted a report to Congress on the potential need for, and technical feasibility of, more stringent (Tier II) motor vehicle tailpipe standards. The Clean Air Act Amendments of 1990 (CAAA) set specific exhaust emission standards, beginning with the 1994 model year for light-duty vehicles and light-duty trucks. These are Tier I standards. The CAAA also requires EPA to study whether further reductions in emissions from these vehicles should be required. These are the Tier II standards, which would not take effect before the 2004 model year. A phase-in would occur between 2004 and 2009, and gradually lead to nearly a full fleet of Tier II compliant vehicles in 2030. This analysis presents estimates of the potential benefits from the Tier II/Gasoline Sulfur rule occurring in 2030.

Chapter 2 describes the methods used to estimate changes in ozone and particulate matter (PM) concentrations and changes in visibility and nitrogen deposition. Chapter 3 describes general issues arising in estimating and valuing changes in adverse health and welfare effects associated with changes in ozone, PM, visibility, and nitrogen deposition. Chapter 4 describes in some detail the methods used for estimating and valuing adverse health effects, while Chapter 5 describes the methods used for welfare effects: crop damage, visibility, nitrogen deposition, and household soiling. The results of these analyses follow in Chapter 6.

This document has three appendices. Appendix A presents the physical and monetary benefits associated with sensitivity calculations for the Tier II 2030 control scenario not considered in the primary analysis. Appendix B presents the ozone C-R functions used in this analysis, and Appendix C presents the PM C-R functions.

2 DEVELOPMENT OF OZONE AND PM AIR QUALITY INPUTS FOR USE IN BENEFITS ANALYSIS

This chapter describes the methods used to forecast changes in ozone, PM, visibility, and nitrogen deposition. Several types of air quality models are used to make these forecasts. In some cases, such as with nitrogen deposition, the model results are ready to be used in the valuation step.[1] In other cases, such as in the case of ozone and PM, we need to carry out a number of steps prior to be able to use these model results. The following sub-sections summarize how air quality model results are used in conjunction with the Criteria Air Pollutant Modeling System (CAPMS) to estimate ozone and PM exposure.

CAPMS is a population-based system for modeling exposures to criteria air pollutants, and is used to estimate health and visibility benefits. CAPMS divides the United States into eight kilometer by eight kilometer grid cells, and estimates the changes in incidence of adverse health and welfare effects associated with given changes in air quality in each grid cell. The national incidence change (or the changes within individual states or counties) is then calculated as the sum of grid-cell-specific changes.

2.1 OZONE AIR QUALITY

To develop baseline and control forecasts for ozone, we use the results of the variable-grid Urban Airshed Model (UAM-V) and observed ozone season data for 1995 and 1996. The modeling data are used to generate "adjustment factors" that quantify the relationship between modeled levels of ozone in the base-year (1995 for the Eastern U.S. and 1996 for the Western U.S.) and the future-year (2030). The adjustment factors are combined with actual monitoring data to generate estimates of the future-year levels of ozone. Note that the modeling data are not used directly (i.e., in an absolute sense) to estimate future-year ozone levels. Instead, we use them in a relative sense to simply adjust actual monitor levels.

For this study, the U.S. was split into an eastern and a western UAM-V modeling region. The eastern region is bounded by longitude -98.5° to -66.5° (roughly east of central South Dakota through central Texas) and latitude 26.33° to 46.67°. Note that small portions of the Eastern U.S. are not covered by the UAM-V modeling (e.g., northern Maine). Thus, in these areas, we assume that ozone levels in the control scenario are identical with those in the baseline scenario. The two simulation periods for the eastern U.S. are based on meteorology for June 12-24 and July 7-15, 1995, and are based on an emission inventory for 1996. The western region is bounded by longitude -126.5° to -98.5° and latitude 26.33° to 51.56°. The two simulation periods for the western U.S. are based on meteorology for July 8-15 and July 21-31, 1996, and are based on an emission inventory for 1996.

We collected ozone monitoring data for the ozone season, defined for this analysis as May through September.[2] An ozone monitor record was considered complete if data were available for 50 percent of days in a given season. Each of these days in turn had to have at least nine hourly observations between 8:00am and 7:59pm.

---

[1] Pechan-Avanti (1999) discuss the estimation of changes in visibility and nitrogen deposition.

[2] EPA has a direct link to the AIRS database: http://www.epa.gov/airs/; however, the data used in this analysis were downloaded from the (password-protected) mainframe version of AIRS, available at: epaibm.rtpnc.epa.gov. Both sets of data are identical; the mainframe allows larger data queries.

In calculating adjustment factors, the UAM-V modeled hourly values from 8:00 am to 7:59 pm are sorted by concentration level for the base-year and the future-year.[3] For each set of modeled data, the ordered hourly values are split evenly into the ten rank-ordered deciles.[4] The average of hourly values in each decile is selected as the representative value for that decile. This means that the first decile's representative ozone level is set equal to the average of values within that decile, and so on for the other deciles. The decile adjustment factors are then calculated as the ratio of the UAM-V future-year scenario's decile to the corresponding UAM-V base-year's decile. Separate decile adjustment factors are calculated for the future baseline and the control scenarios.

We use enhanced Voronoi Neighbor Averaging (eVNA) to interpolate air quality at every population grid cell by first identifying the set of monitors (or pseudo-monitors) that best "surround" the center of the grid cell. Once this set of neighboring monitors is identified for each grid cell, an inverse-distance weight is estimated for each monitor. Using the inverse-distance weights, decile adjustment factors and ozone monitoring data, we calculate hourly ozone values at each CAPMS grid cell in the Eastern U.S. as follows:

$$CAPMS\,cell_{i,j,k,2030} = \left(UAMV_{i,j,2030}\right) \cdot \left(\sum_{h=1}^{N} \frac{mon_{h,j,k,1995}}{UAMV_{h,j,1995}} \cdot d_{h,i} \right)$$

where:

$CAPMS\ cell_{i,j,k,2030}$ = predicted concentration at CAPMS cell *i*, decile group j, hourly observation k

$UAMV_{i,,j,2030}$ = average UAMV modeled 2030 concentration in decile group j of model gridcell closest to CAPMS cell *i*

N = number of neighboring monitors for CAPMS gridcell i

$mon_{h,j,k,1995}$ = observed 1995 ozone level at monitor h, decile group j, hourly observation k

$UAMV_{h,j,1995}$ = average UAMV modeled 1995 concentration in decile group j of model gridcell closest to monitor h

$d_{h,i}$ = inverse-distance weight for cell *i* to monitor h .

Similarly, we calculate ozone forecasts for CAPMS gridcells in the Western U.S. The difference is that we use values for 1996 for the Western U.S., rather than the 1995 values used in the Eastern U.S.

After calculating both baseline and control hourly ozone levels at each CAPMS gridcell, we then calculate the ozone measures that are needed to estimate adverse health effects. For example, a number of studies use the 24-hour daily average ozone level, so for each CAPMS gridcell we get 2030 baseline and control estimates for the 24-hour daily average.

To reduce computational time when estimating the change in health effects associated with daily ozone levels, CAPMS approximates a season's worth of daily ozone measures at each CAPMS gridcell by 20 "bins." Each bin represents five percent of the daily ozone concentrations, and the value for each bin is set at the midpoint of the percentile range it represents. The first bin represents the first (lowest) five percent of the distribution of daily ozone values, and is set at the 2.5th percentile value; the second bin

[3] The data format of Eastern UAM-V modeled hourly output presents all grid cell data starting at 12:00 am., and the Western UAM-V output presents all gridcells starting at 12:00am PST. In processing of data, a correction was encoded to ensure that calculations were based on 8:00 am to 7:59 pm of the appropriate local time zone of the grid cell.

[4]The use of more adjustment factors is generally considered desirable because it provides flexibility; however, it can lead to unreasonably large adjustment factors for lower ozone values, unless a threshold is used (e.g., one ppb as used in this analysis).

represents the next five percent of the distribution of daily values, and is set at the 7.5th percentile value, and so on. Each of the twenty bins therefore represents 7.65 (=153/20) days, since there are 153 days between May and September.

After generating 20 bins for both the baseline and control scenarios, we take the difference between these two values at each bin. We subtract the baseline value in the first bin from the control value in the first bin, and so on for each of the 20 bins. For each CAPMS gridcell, we then get 20 values representing the difference between the baseline and control, and we use these to estimate the change in adverse effects associated with the implementation of the policy. Note that since each value represents 7.65 days, we then multiply each of the 20 incidence change estimates by 7.65 to reconstruct an entire season's worth of incidence changes in the CAPMS grid cell.

2.2 PM AIR QUALITY

We used the results from the Source Receptor (S-R) matrix based on the Climatological Regional Dispersion Model (CRDM) to forecast changes in the ambient concentration of both PM_{10} and $PM_{2.5}$ at the center of each county. Ambient concentrations of PM are composed of directly emitted particles and of secondary aerosols of sulfate, nitrate, and organics. Relative to more sophisticated and resource-intensive three-dimensional modeling approaches, the S-R Matrix does not fully account for all the complex chemical interactions that take place in the atmosphere in the secondary formation of PM.

The S-R Matrix consists of fixed coefficients that reflect the relationship between annual average PM concentration values at a single receptor in each county (i.e., a hypothetical monitor sited at the county population centroid) and the contribution by PM species to this concentration from each emission source in all counties in the 48 contiguous states. The methodology used in this RIA for estimating PM air quality concentrations is detailed in Pechan-Avanti (1999). The following sections describe the steps taken to input these modeled PM levels into CAPMS.

2.2.1 Forecasting PM Based on CRDM

Pechan-Avanti (1999) use the S-R matrix to estimate the 2030 baseline and control scenario mean PM levels, and use regional peak/mean ratios to estimate the peak PM levels for each county in the United States. We then take these mean and peak values to estimate the daily average, annual mean, and annual median PM concentrations that are used in a number of C-R functions.[5] These results are then extrapolated from monitored to unmonitored locations to estimate PM levels at each CAPMS grid-cell based on Voronoi Neighbor Averaging (VNA).

VNA is somewhat different from the eVNA method used to interpolate ozone levels. First, the estimates generated by the S-R matrix are used directly, rather than as a scaling factor that is multiplied with actual ambient PM measures. Second, the model estimates are for each county center, whereas the ozone estimates are generated for UAM-V cells. Third, the interpolation of PM levels to each CAPMS

[5] C-R functions are described in detail in later sections.

gridcell is based on binned data, rather than daily or hourly values.[6] The value for a given bin at a CAPMS gridcell is calculated as follows:

$$CAPMS\,cell_{i,m,2030} = \sum_{h=1}^{N} mon_{h,m,2030} \cdot d_{h,i}$$

where:

$CAPMS\ cell_{i,m,2030}$ = predicted concentration at CAPMS cell *i* for bin m (out of 20 bins)
N = number of neighboring monitors for CAPMS gridcell i
$mon_{h,m,1995}$ = observed 1995 ozone level at monitor h for bin m
$d_{h,i}$ = inverse-distance weight for cell *i* to monitor h .

Once we have estimates for 20 bins for both the baseline and control scenarios, we follow the same procedure that we used with the binned ozone estimates. We take the difference between the baseline and control to estimate the impact of the policy. We subtract the baseline value in the first bin from the control value in the first bin, and so on for each of the 20 bins. For each CAPMS gridcell, we then get 20 values representing the difference between the baseline and control, and we use these to estimate the change in adverse effects associated with the implementation of the policy. Note that since we are interested in PM values for the whole year, each binned value represents 18.25 days (365/20). We then multiply each of the 20 incidence change estimates by 18.25 to reconstruct an entire year's worth of incidence changes in the CAPMS grid cell.

As described below, we develop daily average and the median exposure estimates by first assuming that a gamma distribution is reasonably representative of the PM distribution, and then by using a maximum likelihood estimation procedure to estimate the gamma distribution parameters for each county most consistent with the mean and peak values.[7] A distribution of daily PM values is then estimated for both the baseline and the control scenario in each county, and then the estimated change in PM. This analysis assumes that the order of PM concentrations across days does not change from the baseline to any control scenario, so the change in PM on the n^{th} percentile day equals baseline PM on the n^{th} percentile day minus control scenario PM on the n^{th} percentile day.

Note that for PM_{10}, the peak value is defined as the value corresponding to the 99.7^{th} percentile value of the distribution of actual daily 24-hour average PM_{10} values. For $PM_{2.5}$, the peak value is defined as the value corresponding to the 98^{th} percentile value of the distribution of estimated daily 24-hour average $PM_{2.5}$ values. Also note that daily PM_{10} and $PM_{2.5}$ values derived from the gamma distribution generation procedure are adjusted to reflect the natural occurrence of background concentrations of PM_{10} and $PM_{2.5}$ (the level at which a given PM constituent exists naturally in the environment). Prior to the distribution estimation, an assumed background concentration is subtracted from the mean and peak PM concentrations used to predict the gamma distribution. Once the distribution of daily PM values is predicted, the background concentration is added back to the representative air quality value that has been estimated. In instances where the initial mean value is below a given background concentration assumption, estimates of daily air quality are generated directly from the mean and peak PM values without any background

[6] Recall that in the eVNA method, hourly values were interpolated to each CAPMS gridcell, and the ozone measures of interest were calculated (e.g., 24-hour daily average), then the resulting measures were placed into 20 bins.

[7] We compared a number of different distributions with the distribution of actual PM observations and found the gamma distribution to be most representative.

adjustment. Eastern states are assigned a background threshold of 8ug/m^3 for PM_{10} and 3.5ug/m^3 for $PM_{2.5}$. Western states are assigned a background threshold of 6ug/m^3 for PM_{10} and 2.5ug/m^3 for $PM_{2.5}$.

Estimating the Parameters of a Gamma Distribution, Given the Mean and a Peak Value

The gamma distribution has two parameters, which will be denoted as λ and r, that must be estimated for each county in order for the distribution of daily average PM concentrations to be completely specified. The parameters of a distribution are usually estimated from a random sample drawn from the distribution. Given a sample from the distribution, one of several possible standard methods (for example, maximum likelihood estimation or the method of moments) could be used to estimate the parameters, λ and r. Even given only the sample mean and the sample variance, λ and r could be estimated by the method of moments.

However, neither the whole sample nor the sample variance are available. Instead, the only available information about the distribution is the sample mean and a peak statistic (e.g.,the eighth largest daily average is the 98th percentile point of 365 daily values). The following method, which combines aspects of both the method of moments and maximum likelihood estimation, was therefore used to estimate the two parameters of the gamma distribution from the available statistics.

As in the method of moments, equate the sample mean with the population mean, E(x). The population mean of a gamma distribution is:

$$E(X) = \frac{r}{l} .$$

Therefore, denoting the sample mean as x_s , set:

$$X_S = E(X) = \frac{r}{l} .$$

Solving for λ as a function of x_s and r yields:

$$l = \frac{r}{X_S} .$$

The first piece of information, the sample mean, has been used to reduce the problem from one of estimating two parameters to one of estimating only one parameter. An estimate of r will yield an estimate of λ, given the sample mean.

In the second step, the peak statistic (e.g., the eighth largest daily average PM concentration) is used to estimate r. The distribution of the peak can be derived from the distribution of the daily average PM concentrations.

The peak PM concentration has a probability density function (pdf) that is itself a function of the pdf of the daily PM concentration and the corresponding cumulative distribution function (cdf) of the daily PM concentration. (The cumulative distribution function describes the probability of being less than any

given value.) In particular, if the daily average PM concentration is distributed according to a pdf denoted as f(x; λ, r), and the corresponding cumulative distribution function (cdf) is denoted as F(x; λ, r), then the probability density function of the peak, denoted as $f_{n-\alpha+1}(x;\lambda, r)$, can be shown to be:

$$f_{n-a+1}(x; l, r) = \frac{n!}{(a-1)!(n-a)!}\left[F(x; l, r)\right]^{a-1}\left[1 - F(x; l, r)\right]^{n-a} f(x; l, r) \ ,$$

where n=365 (because there are 365 days in a year) and α represents the peak (e.g., α=358 for the eighth highest $PM_{2.5}$ value out of 365 days)[8]. (Note that the pdf of any order statistic can be derived analogously.) Because λ is a function of r, there is only one unknown parameter that requires estimation.

Maximum likelihood estimation is used to estimate r in the pdf of the peak PM concentration, using the one observation from that pdf -- the peak PM concentration.

The method described above for estimating λ and r has two features that guarantee reasonable estimates. First, the method constrains the estimation of the two parameters so that the estimated population mean, which is a function of both parameters, equals the sample mean. This is reasonable, since the sample mean is the best guess at what the population mean is. Second, this method produces the "most likely" estimate of r, given this constraint. That is, it produces the value of r that maximizes the chance of having gotten the particular second daily maximum PM concentration.

To generate 365 daily PM concentrations from the distribution whose parameters are estimated, we could use Monte Carlo techniques. If the number of iterations in a Monte Carlo exercise is large enough, the frequency distribution of generated observations will approximate the distribution from which the observations were generated. The smaller the number of iterations, however, the rougher the approximation. Instead of generating observations by Monte Carlo techniques, values corresponding to evenly-spaced percentile points of the estimated distribution are used. This guarantees that the sample distribution will correspond to the assumed distribution. First, the percentile of the eighth highest concentration (given) is calculated from the estimated distribution. The percentiles of the 364 other concentrations are evenly spaced around this percentile. The percentile of the highest observation was set midway between the percentile of the second highest observation and the 100th percentile.

Forecasting $PM_{10-2.5}$

The forecast for daily average coarse $PM_{10-2.5}$ (i.e., PM_{10} minus $PM_{2.5}$) is necessary for some C-R functions. To calculate these forecasts, we simply take the difference between the daily PM_{10} and daily $PM_{2.5}$ values for both the baseline and control scenarios. To ensure that coarse PM values remain consistent with both the predicted $PM_{2.5}$ and PM_{10} values, a background concentration adjustment is also applied to coarse $PM_{2.5-10}$ levels. Since coarse PM is equal to the difference between PM_{10} and $PM_{2.5}$, the background threshold for coarse PM is calculated by subtracting $PM_{2.5}$ background concentrations from PM_{10} background concentrations. Eastern coarse PM background is 4.5ug/m3 and Western coarse PM is 3.5ug/m3. Differences between PM_{10} and $PM_{2.5}$ that fall below the background concentration are set to the background level.

[8]The probability density function of the peak is from Mood et al.(1974, p. 254).

3 GENERAL ISSUES IN ESTIMATING HEALTH AND WELFARE BENEFITS

Changes in ozone, PM, nitrogen oxides, and visibility levels result in changes in a number of health and welfare effects, or "endpoints," that society values. This chapter discusses key issues in the estimation of adverse health effects and in the valuation of health and welfare benefits. Section 1 describes general issues that particularly affect the estimation of changes in health effects. Section 2 describes general issues in valuing health and welfare changes. Finally, Section 3 discusses how uncertainty is characterized in this analysis.

3.1 ESTIMATING ADVERSE HEALTH EFFECTS

This section reviews issues that arise in the estimation of adverse health effects. It reviews the derivation of C-R functions, and it reviews how CAPMS combines air quality data and C-R functions. In addition, we discuss how we handle overlapping health effects, thresholds, estimating the baseline incidence rates for the C-R functions, and other issues.

3.1.1 Basic Concentration-Response Model

The methods discussed in this sub-section apply to the estimation of both ozone-related and PM-related changes in adverse health effects. For expository simplicity, the discussion focuses primarily on PM-related changes. The methods, however, are equally applicable to ozone-related changes in effects. Similarly, while several health endpoints have been associated with ozone and PM, the discussion below refers only to a generic "health endpoint," denoted as y. Finally, the discussion refers to estimation of changes in the incidence of the health endpoint at a single location (the population cell, which is equivalent to the CAPMS gridcell). Region-wide changes are estimated by summing the estimated changes over all population cells in the region.

Different epidemiological studies may have estimated the relationship between PM and a particular health endpoint in different locations. The C-R functions estimated by these different studies may differ from each other in several ways. They may have different functional forms; they may have measured PM concentrations in different ways; they may have characterized the health endpoint, y, in slightly different ways; or they may have considered different types of populations. For example, some studies of the relationship between ambient PM concentrations and mortality have excluded accidental deaths from their mortality counts; others have included all deaths. One study may have measured daily (24-hour) average PM concentrations while another study may have used two-day averages. Some studies have assumed that the relationship between y and PM is best described by a linear form (i.e., the relationship between y and PM is estimated by a linear regression in which y is the dependent variable and PM is one of several independent variables). Other studies have assumed that the relationship is best described by a log-linear form (i.e., the relationship between the natural logarithm of y and PM is estimated by a linear regression).[9] Finally, one study may have considered changes in the health endpoint only among members of a particular

[9]The log-linear form used in the epidemiological literature on PM-related health effects is often referred to as "Poisson regression" because the underlying dependent variable is a count (e.g., number of deaths), believed to be Poisson distributed. The model may be estimated by regression techniques but is often estimated by maximum likelihood techniques. The form of the model, however, is still log-linear.

subgroup of the population (e.g., individuals 65 and older), while other studies may have considered the entire population in the study location.

The estimated relationship between PM and a health endpoint in a study location is specific to the type of population studied, the measure of PM used, and the characterization of the health endpoint considered. For example, a study may have estimated the relationship between daily average PM concentrations and daily hospital admissions for "respiratory illness," among individuals age 65 and older, where "respiratory illness" includes International Classification of Disease (ICD) codes A, B, and C.[10] If any of the inputs had been different (for example, if the entire population had been considered, or if "respiratory illness" had consisted of a different set of ICD codes), the estimated C-R function would have been different. When using a C-R function estimated in an epidemiological study to estimate changes in the incidence of a health endpoint corresponding to a particular change in PM in a population cell, then, it is important that the inputs be appropriate for the C-R function being used -- i.e., that the measure of PM, the type of population, and the characterization of the health endpoint be the same as (or as close as possible to) those used in the study that estimated the C-R function.

Estimating the relationship between PM and a health endpoint, y, consists of (1) choosing a functional form of the relationship and (2) estimating the values of the parameters in the function assumed. The two most common functional forms in the epidemiological literature on PM (and ozone) and health effects are the log-linear and the linear relationship. The log-linear relationship is of the form:

$$y = Be^{b \cdot PM} \ ,$$

or, equivalently,

$$ln(y) = a + b \cdot PM \ ,$$

where the parameter B is the incidence of y when the concentration of PM is zero, the parameter β is the coefficient of PM, ln(y) is the natural logarithm of y, and $\alpha = \ln(B)$.[11] If the functional form of the C-R relationship is log-linear, the relationship between ΔPM and Δy is:

$$\Delta y = y \cdot \left(e^{b \cdot \Delta PM} - 1\right) \ ,$$

where y is the baseline incidence of the health effect (i.e., the incidence before the change in PM). For a log-linear C-R function, the relative risk (RR) associated with the change ΔPM is:

[10] The International Classification Codes are described at the website of the Medical Center Information Systems: Duke University Health Systems (1999).

[11] Other covariates besides pollution clearly affect mortality. The parameter B might be thought of as containing these other covariates, for example, evaluated at their means. That is, $B = B_o \exp\{\beta_1 x_1 + ... + \beta_n x_n\}$, where B_o is the incidence of y when all covariates in the model are zero, and $x_1, ... , x_n$ are the other covariates evaluated at their mean values. The parameter B drops out of the model, however, when changes in incidences are calculated, and is therefore not important.

$$RR_{\Delta PM} = e^{b \cdot \Delta PM} \quad .$$

Epidemiological studies often report a relative risk for a given ΔPM, rather than the coefficient, β, in the C-R function. The coefficient can be derived from the reported relative risk and ΔPM, however, by solving for β:

$$b = \frac{\ln(RR)}{\Delta PM} \quad .$$

The linear relationship is of the form:

$$y = a + b \cdot PM \quad ,$$

where α incorporates all the other independent variables in the regression (evaluated at their mean values, for example) times their respective coefficients. When the C-R function is linear, the relationship between a relative risk and the coefficient, β, is not quite as straightforward as it is when the function is log-linear. Studies using linear functions usually report the coefficient directly.

If the functional form of the C-R relationship is linear, the relationship between ΔPM and Δy is simply:

$$\Delta y = b \cdot \Delta PM \quad .$$

A few epidemiological studies, estimating the relationship between certain morbidity endpoints and PM, have used functional forms other than linear or log-linear forms. Of these, logistic regressions are the most common. Abt Associates (1999, Appendix A) provides further details on the derivation of dose-response functions.

3.1.2 Calculation of Adverse Health Effects with CAPMS

CAPMS is a population-based system for modeling exposure to ambient levels of criteria air pollutants and estimating the adverse health effects associated with this exposure. CAPMS divides the United States into multiple grid cells, and estimates the changes in incidence of adverse health and welfare effects associated with given changes in air quality in each grid cell. The national incidence change (or the changes within individual states or counties) is then calculated as the sum of grid-cell-specific changes.

To calculate point estimates of the changes in incidence of a given selection of adverse health and welfare effects associated with a given set of air quality changes, CAPMS goes through the following steps at each CAPMS grid cell:

- Interpolate the air quality in the baseline scenario and in the control scenario at the CAPMS grid cell center, as described in Chapter 2. If the daily values have been binned at the monitors from

which the interpolation is carried out, the resulting baseline and control scenario air quality data at the CAPMS grid cell center is also binned.

- Calculate the changes in air quality from baseline to control scenario in the CAPMS grid cell. The changes in air quality are calculated as the differences between the baseline bins and the corresponding control scenario bins. The change in the n^{th} bin concentration is the difference between the baseline n^{th} bin concentration and the control scenario n^{th} bin concentration.

- Access the selected C-R functions being used, and the required baseline incidence rates and grid cell population.

- Using the above inputs, calculate the change in incidence of each adverse health effect for which a C-R function has been accessed.

For functions based on changes in daily average pollutant concentrations, estimated incidence changes corresponding to air quality changes in each of the 20 bins are summed. This summed incidence, however, is the result of 20 representative air quality changes (one for each bin). Recall that each bin represents 18.25 days for PM (to represent a year's worth of exposure) and 7.65 days for ozone (to represent an ozone season's worth of exposure). To adjust the summed incidence estimate, it is multiplied by either 18.25 to produce an annual change, or by 7.65 to produce a seasonal change. This procedure is applied to each grid cell in CAPMS. The resulting incidence change is stored, and CAPMS proceeds to the next grid cell, where the above process is repeated. The national change (or the change in any designated geographical area) is calculated at the end of the process by summing the grid cell-specific changes.

To reflect the uncertainty surrounding predicted incidence changes resulting from the uncertainty surrounding the pollutant coefficients in the C-R functions used, CAPMS produces a *distribution* of possible incidence changes for each adverse health, rather than a single point estimate. To do this, it uses both the point estimate of the pollutant coefficient (β in the above equation) and the standard error of the estimate to produce a normal distribution with mean equal to the estimate of β and standard deviation equal to the standard error of the estimate. Using a Latin Hypercube method,[12] we take the n^{th} percentile value of β from this normal distribution, for n = 0.5, 1.5, ..., 99.5, and follow the procedure outlined in the section above to produce an estimate of the incidence change, given the β selected. Repeating the procedure for each value of β selected results in a distribution of incidence changes in the CAPMS grid cell. This distribution is stored, and CAPMS proceeds to the next grid cell, where the process is repeated. A distribution of the national change (or change in a designated geographical area) is calculated by summing the n^{th} percentile grid cell-specific changes, for n = 0.5, 1.5, ..., 99.5.

3.1.3 Population Projections

Benefits for the Tier II analysis are based on health and welfare effect incidence changes due to predicted air quality improvements in the year 2030. Integral to the estimation of such benefits is an accurate estimate of future population projections. Though similar benefits analyses have preceded this

[12]The Latin Hypercube method is used to enhance computer processing efficiency. It is a sampling method that divides a probability distribution into intervals of equal probability, with an assumption value for each interval assigned according to the interval's probability distribution. Compared with conventional Monte Carlo sampling, the Latin Hypercube approach is more precise over a fewer number of trials because the distribution is sampled in a more even, consistent manner (Decisioneering, 1996, pp. 104-105).

one, using population projections out to various future years, no analysis has been conducted out to the year 2030. This section describes the method used to estimate county-level 2030 populations.

The underlying data used to create county-level 2030 population projections is based on: (1) 1990 county-level population statistics for all U.S. counties collected by the U.S. Census (Wessex, 1994), and (2) future-year state and metropolitan area population estimates provided by the Bureau of Economic Analysis (1995). Growth factors are calculated using the BEA data and are applied to the 1990 county-level populations.

A growth factor is calculated by taking the ratio of an estimated region's 2030 population divided by the1990 population for that same area. Population estimates for the years 1990-93, 2000, 2005, 2010, 2015, 2025 and 2045 were collected by the BEA. A 2030 population estimate was not provided. Instead, 2030 state and metropolitan area populations were interpolated linearly using estimates from the years 2025 and 2045.

Growth factors are calculated for both urban areas and rural areas. An urban area is defined as a county that falls within one of the metropolitan areas for which the beapop file contains population data. This includes metropolitan statistical areas (MSAs), primary metropolitan statistical areas (PMSAs), consolidated metropolitan statistical areas (CMSAs), and New England county metropolitan areas (NECMAs) (as defined by U.S. Census Bureau, 1999).[13] In this section, however, all metropolitan areas are referred to as MAs. A rural area is defined as a county that falls outside the defined metropolitan areas.

Urban areas grow according to the growth rate calculated for the particular metropolitan area within which they are located. This adjustment is very straightforward, simply taking the ratio of future year to base year metropolitan area population and multiplying that factor by the base year county population. The equation is:

$$2030CountyPop_i = 1990CountyPop_i \cdot \frac{2030\,MAPop_i}{1990\,MAPop_i}$$

where:

$2030CountyPop_i$ = projected 2030 population in urban county i
$1990CountyPop_i$ = actual 1990 population for county i
$2030MAPop_i$ = projected 2030 population in metropolitan area for county i
$1990MAPop_i$ = actual 1990 population for metropolitan area for county i.

Rural areas grow according to the growth rate calculated for the particular state within which they are located, adjusted to subtract out metropolitan area populations. Before the ratio of future year to base year state population is calculated, the population attributed to all metropolitan areas located within that state is subtracted from the future year and base year population totals. Once this metropolitan area adjustment has been made, the rural growth factor is multiplied by the base-year population in all non-MA counties to get future-year population projections. The equation is:

[13] The Census Bureau definitions are available at: http://www.census.gov/population/www/estimates/aboutmetro.html .

$$2030CountyPop_i = 1990CountyPop_i \cdot \frac{(2030StatePop_i - \sum 2030MAPop_i)}{(1990StatePop_i - \sum 1990MAPop_i)}$$

where:

$2030CountyPop_i$ = projected 2030 population in rural county i
$1990CountyPop_i$ = actual 1990 population for county i
$2030StatePop_i$ = projected 2030 population in state where county i is located
$1990State\ Pop_i$ = actual 1990 population for state where county i is located
$\Sigma 2030MAPop_i$ = projected 2030 population in metropolitan areas located in state with county i
$\Sigma 1990MAPop_i$ = actual 1990 population for metropolitan areas located in state with county i .

One problem that exists with this method is that many metropolitan areas cross state boundaries. To accurately subtract urban populations from state populations, we need to know the urban county populations for both 1990 and 2030. Using the county populations for 1990, we can estimate the portion of a particular metropolitan area's population that belongs to a given state. However, we do not have 2030 county population projections with which to apportion 2030 metropolitan area populations. To remedy this, we apply the same percent of the population a given county contributes to a metropolitan area in 1990 to 2030 metropolitan areas when apportioning populations between states.

3.1.4 Overlapping Health Effects

Several endpoints reported in the health effects literature overlap with each other. Hospital admissions for single respiratory ailments (e.g. pneumonia) overlap with estimates of hospital admissions for "all respiratory" ailments.[14] Similarly, several studies quantify the occurrence of respiratory symptoms where the definitions of symptoms are not unique (e.g., shortness of breath or upper respiratory symptoms). In choosing studies to include in the aggregated benefits estimate (discussed below), this analysis carefully considers the issue of double-counting benefits that might arise from overlapping health effects.

3.1.5 Baseline Incidences

As noted above, most of the relevant C-R functions are log-linear, and the estimation of incidence changes based on a log-linear C-R function requires a baseline incidence. The baseline incidence for a given CAPMS population cell is the baseline incidence rate in that location multiplied by the relevant population. County mortality rates are used in the estimation of air pollution-related mortality, and all CAPMS population cells in the county are assumed to have the same mortality rate. Hospital admissions are only available at the national level, so all areas are assumed to have the same incidence rate for a given population age group. For some endpoints, such as respiratory symptoms and illnesses and restricted activity days, baseline incidence rates are not available even at the national level. The only sources of estimates of baseline incidence rates in such cases are the studies reporting the C-R functions for those health endpoints. The baseline incidence rate and its source are given for each C-R function in Appendices B and C.

[14]Pneumonia is often classified with the International Classification of Diseases (ICD) codes of 480-486, while all respiratory admissions are classified with ICD codes 460-519.

3.1.6 Thresholds

A very important issue in applied modeling of changes in PM is whether to apply the C-R functions to all predicted changes in ambient concentrations, even small changes occurring at levels approaching the concentration in which they exist in the natural environment (without interference from humans), referred to as "anthropogenic background." Different assumptions about whether to model thresholds, and if so, at what levels, can have a major effect on the resulting benefits estimates.[15]

None of the epidemiological functions relating PM to various health and welfare endpoints incorporate thresholds. Instead, all of these functions are continuous and differentiable down to zero pollutant levels. A threshold may be imposed on these models, however, in several ways, and there are various points at which the threshold could be set. (A threshold can be set at any point. There are some points, however, that may be considered more obvious candidates than others.) One possible threshold might be the background level of the pollutant. Another might be a relevant standard for the pollutant. Whatever the threshold, the implication is that there are no effects below the threshold.

A threshold model can be constructed in more than one way. One method is to simply truncate the C-R function at the threshold (i.e., to not include any physical effect changes associated with PM concentrations below the designated threshold). This method uses the original C-R function, but calculates the change in PM as [max(T,baseline PM) - max(T, regulatory alternative PM)], where T denotes the designated threshold. This threshold model will predict a smaller incidence of the health effect than the original model without a threshold. Clearly, as T increases, the predicted incidence of the health effect will decrease.

An alternative method is to replace the original C-R function with a "hockey stick" model that best approximates the original function that was estimated using actual data. The hockey stick model is horizontal up to a designated threshold PM level, T, and is linear with a positive slope for PM concentrations greater than T. Recall the log-linear C-R function:

$$y = a + b \cdot PM \ .$$

Assuming that the value of the coefficient, β, depends on the level of PM, we get:

$$\ln(y) = a' \ , \quad for\ PM \le T \ , and$$
$$\ln(y) = a' + b' \cdot PM \ , \quad for\ PM > T \ .$$

Ideally, the coefficients would be estimated based on the data in the original study – that is, a hockey stick model would be fit to the original data, so that the threshold model that is most consistent with the available information would be chosen. If a threshold model could be estimated from the original data, it is unlikely that α' would equal α or that β' would equal β, because such a hockey stick model would be consistently below the original model, except at PM=0 (where the two models would coincide). If that were the hockey stick model that best fit the data, then it is unlikely that the best fitting linear model would be consistently above it. Instead, the hockey stick model that best fits the same data would most likely have α'>α and β'>β. A graph of this model would therefore cross the graph of the linear model at two points.

[15]Thresholds may also apply to ozone, however, recent RIAs have not explicitly modeled ozone thresholds.

Whether such a hockey stick threshold model predicted a greater or smaller incidence of the health effect than the linear model would depend on the distribution of PM levels. It is worth noting that the graph of the first type of threshold model, in which the C-R function is simply truncated at the threshold, would be discontinuous at the threshold. This is highly unlikely to be a good model of the actual relationship between PM and any health endpoint.

There is some evidence that, at least for particulate matter, not only is there no threshold, but the PM coefficient may actually be larger at lower levels of PM and smaller at higher levels. Examining the relationship between particulate matter (measured as TSP) and mortality in Milan, Italy during the ten year period 1980-1989, Rossi et al. (1999) fitted a model with one slope across the entire range of TSP and an additional slope for TSP greater than 200 $\mu g/m^3$. The second slope was statistically significant ($p<0.0001$) and negative, indicating a lower slope at higher TSP levels.

3.1.7 Application of a Single C-R Function Everywhere

Whether the C-R relationship between a pollutant and a given health endpoint is estimated by a single function from a single study or by a pooled function of C-R functions from several studies, that same C-R relationship is applied everywhere in the benefits analysis. Although the C-R relationship may in fact vary somewhat from one location to another (for example, due to differences in population susceptibilities or differences in the composition of PM), location-specific C-R functions are available only for those locations in which studies were conducted. While a single function applied everywhere may result in overestimates of incidence changes in some locations and underestimates of incidence changes in other locations, these location-specific biases will to some extent cancel each other out when the total incidence change is calculated. It is not possible to know the extent or direction of the bias in the total incidence change based on application of a single C-R function everywhere.

3.1.8 Estimating Pollutant-Specific Benefits Using Single Pollutant vs. Multi-Pollutant Models

Many studies include both ozone and particulate matter in their final models. It is often difficult to separate out the effect of a single pollutant from the effects of other pollutants in the mix. Multi-pollutant models have the advantage that the coefficient for a single pollutant in such a model will be unbiased (so that the effects of other pollutants will not be attributed falsely to the single pollutant). However, the variance of the estimator of the coefficient of the pollutant of interest will increase as the correlations between the other pollutants in the model and that pollutant increase. If the other pollutants in the model are highly correlated with the pollutant of interest, we would have an unbiased but unstable (high variance) estimator. However, while single pollutant models have the advantage of more stable estimators, the coefficient estimate in a single pollutant model could be biased in such a model. We could consider the single pollutant as an "indicator pollutant" – i.e., an indicator of a pollution mix – if we use single pollutant models. However, there is no guarantee that the composition of the pollution mix will remain the same under a control scenario that targets only a single pollutant.

This analysis uses both single pollutant and multi-pollutant models to derive pollutant-specific benefits estimates. When more than one study has estimated the relationship between a given endpoint and a given pollutant, information from both single-pollutant and multi-pollutant models may be pooled to derive pollutant-specific benefits estimates. For example, the benefits predicted by a model with only PM may be pooled with the benefits predicted by a model with both PM and ozone to derive an estimate of the PM-related benefits associated with a given endpoint. If the benefits of PM-related and ozone-related

incidence changes are both being calculated and added together, there is the possibility of overestimating benefits if some of the studies used are single pollutant models. Suppose, for example, that only ozone is actually associated with a given endpoint, but PM appears to be associated only because it is correlated with ozone. The benefits predicted by a single pollutant PM model would, in that case, actually reflect the benefits of reducing ozone, to the extent that PM and ozone are correlated. If those "PM-related" benefits were then added to the ozone-related benefits calculated from other models, a likely result would be the overstatement of benefits of reducing ozone. If only one pollutant is being associated with the endpoint in this analysis (e.g., chronic bronchitis is associated only with PM in this analysis, while chronic asthma is associated only with ozone), this is not a problem.

3.1.9 Pooling Study Results

When only a single study has estimated the C-R relationship between a pollutant and a given health endpoint, the estimation of a population cell-specific incidence change, Δy, is straightforward, as noted above. When several studies have estimated C-R relationships between a pollutant and a given health endpoint, the results of the studies can be pooled to derive a single estimate of the function. If the functional forms, pollutant averaging times, and study populations are all the same (or very similar), a pooled, "central tendency" C-R function can be derived from multiple study-specific C-R functions. Even if there are differences among the studies, however, that make a pooled C-R function infeasible, a pooled estimate of the incidence change, Δy, and/or the monetary benefit of the incidence change can be obtained by incorporating the appropriate air quality data into the study-specific C-R functions and pooling the resulting study-specific predictions of incidence change. Similarly, study-specific predictions of incidence change can be combined with unit dollar values to produce study-specific predictions of benefits.

Whether the pooling is done in "coefficient space," "incidence change space," or "dollar space," the question of the relative weights assigned to the estimates (of coefficients, incidence changes, or dollar benefits) from each input study must be addressed. One possibility is simply averaging the estimates from all the studies. This has the advantage of simplicity, but the disadvantage of not taking into account the measured uncertainty of each of the estimates. Estimates with great uncertainty surrounding them are given the same weight as estimates with very little uncertainty.

An alternative approach to pooling incidence estimates from different studies is to give more weight to studies with little estimated variance than to studies with a great deal of estimated variance. The exact way in which weights are assigned to estimates from different studies in a pooled analysis depends on the underlying assumption about how the different estimates are related to each other. Under the assumption that there is actually a distribution of true effect coefficients, or β's, that differ by location and/or study (referred to as the random effects model), the different coefficients reported by different studies may be estimates of *different* underlying coefficients, rather than just different estimates of the same coefficient. In contrast to the "fixed-effects" model (which assumes that there is only one β everywhere), the random-effects model allows the possibility that different studies are estimating different parameters.[16]

[16] In studies of the effects of PM_{10} on mortality, for example, if the composition of PM_{10} varies among study locations the underlying relationship between mortality and PM_{10} may be different from one study location to another. For example, fine particles make up a greater fraction of PM_{10} in Philadelphia County than in Southeast Los Angeles County. If fine particles are disproportionately responsible for mortality relative to coarse particles, then one would expect the true value of β for PM_{10} in Philadelphia County to be greater than the true value of β for PM_{10} in Southeast Los Angeles County. This would violate the assumption of the "fixed effects" model. However, applying a random effects model assumes that the observed set of coefficients in the policy region.

A third approach to pooling studies is to apply subjective weights to the studies, rather than conducting a random effects pooling analysis. If the analyst is aware of specific strengths and weaknesses of the studies involved, this prior information may be used as input to the calculation of weights which reflect the relative reliability of the estimates from the studies.

In those cases in which pooling of information from multiple studies was an option in this analysis, pooling was done in both "incidence change space" and "dollar benefit space." The hypothesis of fixed effects was tested. If this hypothesis was rejected, an underlying random effects model was used as the basis for weighting of studies. A more detailed description of the pooling procedure used is given below in the section on hospital admissions.

3.2 VALUING CHANGES IN HEALTH AND WELFARE EFFECTS

This section discusses a number of issues that arise in valuing changes in health and welfare effects. The first section provides some background on willingness to pay (WTP). The second section discusses the possibility that as income changes then WTP would also change. The third section describes how WTP estimates, that were originally calculated in 1990 dollars, are corrected for inflation to get estimates in 1997 dollars. In the last section, we briefly review how we aggregate benefits estimates.

3.2.1 WTP Estimation

WTP is a measure of value an individual places on gaining an outcome viewed as desirable, be it something that can be purchased in a market or not. The WTP measure, therefore, is the amount of money such that the individual would be indifferent between having the good (or service) and having the money. An alternative measure of economic value is willingness to accept (WTA) a monetary compensation to offset a deterioration in welfare, such that the individual would be indifferent between having the money and not having the deterioration. Whether WTP or WTA is the appropriate measure depends on how property rights are assigned. Consider an increase in air pollution. If society has assigned property rights so that people have a right to clean air, then they must be compensated for an increase in the level of air pollution. The appropriate measure of the value of avoiding an increase in air pollution, in this case, would be the amount people would be willing to accept in compensation for the more polluted air. If, on the other hand, society has not assigned people the right to clean air, then the appropriate measure of the value of avoiding an increase in air pollution would be what people are willing to pay to avoid it. The assignment of property rights in our society is unclear. WTP is by far the more common measure used in benefits analyses, however, reflecting the fact that this is a much more common measure in the empirical valuation literature. In this analysis, wherever possible, the valuation measures are in terms of WTP. Where such estimates are not available, alternative measures are used, such as cost-of-illness and wage-risk studies. These are discussed for each endpoint where applicable.

For both market and non-market goods, WTP reflects individuals' preferences. Because preferences are likely to vary from one individual to another, WTP for both market (e.g., the purchase of a new automobile) and non-market goods (e.g., health-related improvements in environmental quality) is likely to vary from one individual to another. In contrast to market goods, however, non-market goods, such as environmental quality improvements, are public goods whose benefits are shared by many individuals. The individuals who benefit from the environmental quality improvement may have different WTPs for this non-market good. The total social value of the good is the sum of the WTPs of all individuals who "consume" (i.e., benefit from) the good.

In the case of health improvements related to pollution reduction, it is not certain specifically who will receive particular benefits of reduced pollution. For example, the analysis may predict 100 hospital admissions for respiratory illnesses avoided, but the analysis does not estimate which individuals will be spared those cases of respiratory illness that would have required hospitalization. The health benefits conferred on individuals by a reduction in pollution concentrations are, then, actually *reductions in the risk* of having to endure certain health problems. These benefits (reductions in risk) may not be the same for all individuals (and could be zero for some individuals). Likewise, the WTP for a given benefit is likely to vary from one individual to another. In theory, the total social value associated with the decrease in risk of a given health problem resulting from a given reduction in pollution concentrations is:

$$\sum_{i=1}^{N} WTP_i\left(B_i\right) ,$$

where B_i is the benefit (i.e., the reduction in risk of having to endure the health problem) conferred on the i^{th} individual (out of a total of N) by the reduction in pollution concentrations, and $WTP_i(B_i)$ is the i^{th} individual's WTP for that benefit.

If a reduction in pollution concentrations affects the risks of several health endpoints, the total health-related social value of the reduction in pollution concentrations is:

$$\sum_{i=1}^{N}\sum_{j=1}^{J} WTP_i\left(B_{i,j}\right) ,$$

where B_{ij} is the benefit related to the j^{th} health endpoint (i.e., the reduction in risk of having to endure the j^{th} health problem) conferred on the i^{th} individual by the reduction in pollution concentrations, and $WTP_i(B_{ij})$ is the i^{th} individual's WTP for that benefit.

The reduction in risk of each health problem for each individual is not known, nor is each individual's WTP for each possible benefit he or she might receive known. Therefore, in practice, benefits analysis estimates the value of a *statistical* health problem avoided. For example, although a reduction in pollutant concentrations may save actual lives (i.e., avoid premature mortality), whose lives will be saved cannot be known *ex ante*. What is known is that the reduction in air pollutant concentrations results in a reduction in mortality risk. It is this reduction in mortality risk that is valued in a monetized benefit analysis. Individual WTPs for small reductions in mortality risk are summed over enough individuals to infer the value of a *statistical* life saved. This is different from the value of a particular, identified life saved. Rather than "WTP to avoid a death," then, it is more accurate to use the term "the value of a statistical life."

Suppose, for example, that a given reduction in PM concentrations results in a decrease in mortality risk of 1/10,000. Then for every 10,000 individuals, one individual would be expected to die in the absence of the reduction in PM concentrations (who would not die in the presence of the reduction in PM concentrations). If WTP for this 1/10,000 decrease in mortality risk is $500 (assuming, for now, that all individuals' WTPs are the same), then the value of a statistical life is 10,000 x $500, or $5 million.

A given reduction in PM concentrations is unlikely, however, to confer the same risk reduction (e.g., mortality risk reduction) on all exposed individuals in the population. (In terms of the expressions above, B_i is not necessarily equal to B_j , for $i \neq j$). In addition, different individuals may not be willing to pay the same amount for the same risk reduction. The above expression for the total social value associated with the decrease in risk of a given health problem resulting from a given reduction in pollution concentrations may be rewritten to more accurately convey this. Using mortality risk as an example, for a given unit risk reduction (e.g., 1/1,000,000), the total mortality-related benefit of a given pollution reduction can be written as:

$$\sum_{i=1}^{N} \int_{0}^{n_i} marginal\ WTP_i(x)dx \ ,$$

where marginal $WTP_i(x)$ is the i^{th} individual's marginal willingness to pay curve, n_i is the number of units of risk reduction conferred on the i^{th} exposed individual as a result of the pollution reduction, and N is the total number of exposed individuals.

The values of a statistical life implied by the value-of-life studies were derived from specific risk reductions. Implicit in applying these values to a situation involving possibly different risk reductions is the assumption that the marginal willingness to pay curve is horizontal – that is, that WTP for n units of risk reduction is n times WTP for one unit of risk reduction. If the marginal willingness to pay curve is horizontal, the integral in the above expression becomes a simple product of the number of units of risk reduction times the WTP per unit. The total mortality-related benefit (the expression above) then becomes:

$$\sum_{i=1}^{N} \left(number\ of\ units\ of\ risk\ reduction\right)_i \cdot \left(\frac{WTP_i}{unit\ of\ risk\ reduction} \right).$$

If different subgroups of the population have substantially different WTPs for a unit risk reduction and substantially different numbers of units of risk reduction conferred on them, then estimating the total social benefit by multiplying the population mean WTP (MWTP) to save a statistical life times the predicted number of statistical lives saved could yield a biased result. Suppose, for example, that older individuals' WTP per unit risk reduction is less than that of younger individuals (e.g., because they have fewer years of expected life to lose). Then the total benefit will be less than it would be if everyone's WTP were the same. In addition, if each older individual has a larger number of units of risk reduction conferred on him (because a given pollution reduction results in a greater absolute reduction in risk for older individuals than for younger individuals), this, in combination with smaller WTPs of older individuals, would further reduce the total benefit.

While the estimation of WTP for a market good (i.e., the estimation of a demand schedule) is not a simple matter, the estimation of WTP for a non-market good, such as a decrease in the risk of having a particular health problem, is substantially more difficult. Estimation of WTP for decreases in very specific health risks (e.g., WTP to decrease the risk of a day of coughing or WTP to decrease the risk of admission to the hospital for respiratory illness) is further limited by a paucity of information.[17] Derivation of the dollar value estimates discussed below was often limited by available information.

[17] Some health effects, such as technical measures of pulmonary functioning (e.g., forced expiratory volume in one second) are frequently studied by epidemiologists, but there has been very little work by economists on valuing these changes (e.g., Ostro et al., 1989a).

3.2.2 Change Over Time in WTP in Real Dollars

The WTP for health-related environmental improvements (in real dollars) could change between now and the year 2030. If real income increases between now and the year 2030, for example, it is reasonable to expect that WTP, in real dollars, would also increase. Below we summarize the evidence regarding this effect, however we do not adjust our results in this analysis, because of the uncertainty regarding the size of the effect.

Based on historical trends, the U.S. Bureau of Economic Analysis projects that, for the United States as a whole as well as for regions and states within the U.S., mean per capita real income will increase. For the U.S. as a whole, for example, mean per capita personal income is projected to increase by about 16 percent from 1993 to 2005 (U.S. Bureau of Economic Analysis, 1995).

The mean WTP in the population is the correct measure of the value of a health problem avoided, and that WTP is a function of income. If the mean per capita real income rises by the year 2030, the mean WTP would probably rise as well. While this is most likely true, the degree to which mean WTP rises with a rise in mean per capita income is unclear unless the elasticity of WTP with respect to changes over time in real income is 1.0.

There is some evidence (Alberini et al., 1997; Loehman and De, 1982; Mitchell and Carson, 1986) that the elasticity of WTP for health-related environmental improvements with respect to real income is less than 1.0, possibly substantially so. If this is the case, then changes in mean income cannot be readily translated into corresponding changes in mean WTP. Although an increase in mean income is likely to imply an increase in mean WTP, the degree of the increase cannot be ascertained from information only about the means.

Several factors, in addition to real income, that could affect the estimated benefit associated with reductions in air pollution concentrations could also change by the year 2030. Demographic characteristics of exposed populations could change. Technological advances could change both the nature of precursor emissions to the ambient air and the susceptibility of individuals to air pollution. Any such changes would be reflected in C-R functions that differ from those that describe current relationships between ambient concentrations and the various health endpoints. While adjustments of WTP to reflect changes in real income are of interest, such adjustments would by no means necessarily reflect all possible changes that could affect the benefits of reduced air pollution in 2030.

3.2.3 Adjusting Benefits Estimates from 1990 Dollars to 1997 Dollars

This section describes the methods used to convert benefits estimates into constant dollars. In past RIA analyses, cost and benefit estimates have been presented in constant 1990 dollars. Benefits estimates in this analysis, however, are presented in constant 1997 dollars. To adjust benefits estimates from 1990 dollars to 1997 dollars, the method of adjustment depends on the basis of the benefits estimates. These methods are presented below. Four different bases of estimates are delineated in Exhibit 3-1, including that for agricultural benefits.[18]

[18]Agricultural benefits are discussed in Chapter 3.

Exhibit 3-1 Bases of Benefits Estimation

Basis of Benefit Estimation	Benefit Endpoints
Cost of illness	Hospital admissions avoided
Direct estimates of WTP	Statistical lives saved; statistical life-years saved Chronic bronchitis; chronic asthma Morbidity endpoints using WTP Visibility -- residential Visibility -- recreational Consumer cleaning cost savings
Earnings	Work loss days (WLDs) avoided Increased worker productivity
Changes in yields and prices of market commodities	Agricultural benefits

Benefits estimates based on cost-of-illness have been adjusted by using the consumer price indexes (CPI-Us) for medical care. Because increases in medical costs have been significantly greater than the general rate of inflation, using a general inflator (the CPI-U for "all items" or some other general inflator) to adjust from 1990 to 1997 dollars would downward bias cost-of-illness estimates in 1997 dollars.

Benefits estimates based directly on estimates of WTP have been adjusted using the CPI-U for "all items." (The CPI-Us, published by the U.S. Dept. of Labor, Bureau of Labor Statistics, can also be found in Council of Economic Advisers (e.g.1997)) An overview of the adjustments from 1990 to 1997 dollars for WTP-based and cost-of-illness based valuations is given in Exhibit 3-2.

Exhibit 3-2 Consumer Price Indexes Used to Adjust WTP-Based and Cost-of-Illness-Based Benefits Estimates from 1990 Dollars to 1997 Dollars

	1990 (1)	1997 (2)	Adjustment Factor [a] (2)/(1)	Relevant Endpoints
CPI-U for "All Items" [b]	130.7	160.5	1.228	WTP-based valuation: 1. Statistical lives saved [c] 2. Chronic bronchitis; chronic asthma 3. Morbidity endpoints using WTP [d] 3. Visibility -- residential 4. Visibility -- recreational 5. Consumer cleaning cost savings
CPI-U for Medical Care [b]	162.8	234.6	1.441	Cost-of-illness based valuation: Hospital admissions avoided [e]

[a] Benefits estimates in 1990 dollars are multiplied by the adjustment factor to derive benefits estimates in 1997 dollars.

[b] Source: Dept. of Labor, Bureau of Labor Statistics; reported in Council of Economic Advisers (1998, Table B-60)

[c] Adjustments to 1990 $ were originally made by Industrial Economics Inc. using the CPI-U for "all items" (IEc1992).

[d] Adjustments of WTP-based benefits for morbidity endpoints to 1990 $ were originally made by Industrial Economics Inc. (1993) using the CPI-U for "all items."

[e] Adjustments of cost-of-illness based estimates of all hospital admissions avoided to 1990 $ were made by Abt Associates Inc. in previous analyses, such as the NAAQS RIA (U.S. EPA, 1997c).

Benefits estimates for two endpoints, work loss days (WLDs) avoided and increased worker productivity, have in past analyses been based on the mean or median daily wage. Consistent with economic welfare theory, the valuation of benefits associated with increased worker productivity resulting from improved ozone air quality used the average daily income for outdoor workers engaged in strenuous activity, reported by the 1990 U.S. Census ($73 per day, in 1990). The valuation of the benefit of avoiding a work loss day used the median daily income rather than the mean. The income distribution in the United States is highly skewed, so that the mean income is substantially larger than the median income. However, the incomes of those individuals who lose work days due to pollution are not likely to be a random sample from this income distribution. In particular, the probability of being drawn from the upper tail of the distribution is likely to be substantially less than the probability mass in that tail. To reflect this likelihood, we used the median income rather than the mean income as the value of a work loss day. This is explained more fully below in the section on valuing work loss days.

The benefits estimates for WLDs avoided and for increases in worker productivity can be put into 1997 dollars in several ways. The most straightforward approach for WLDs is to obtain the 1997 median weekly earnings (and divide by five to derive the median daily earnings) rather than relying on adjustments from 1990 to 1997 dollars. The median weekly earnings of full-time wage and salary workers in 1997 was $503 (U.S. Bureau of the Census 1998, Table 696). This implies a median daily earnings of $100.6, or rounded to the nearest dollar, $101. Alternatively, we can adjust the median daily wage for 1990 to 1997 dollars, using the CPI-U for "all items." The result turns out to be the same. The adjustment factor (the ratio of the 1997 CPI-U to the 1990 CPI-U) is 1.228. Applied to the median daily earnings of $82.4 in 1990, the median daily earnings in 1997 would be $101.2, or rounded to the nearest dollar, $101.

The simplest method to adjust the benefits estimate for increased worker productivity would be to use the CPI-U for "all items" to adjust the current estimate of $73 per day, in 1990 dollars, to 1997 dollars. This would result in an estimate of $73*1.228 = $89.6 per day, or rounded to the nearest dollar, $90 per day, in 1997 dollars. Alternatively, we could try to obtain an estimate of the average daily income for outdoor workers engaged in strenuous activity in 1997, as we previously did for 1990. It is not entirely clear, however, which categories of workers were included among "outdoor workers engaged in strenuous activity" to obtain the 1990 estimate of $73 per day. It is therefore not clear which categories to include to derive an equivalent figure for 1997.

Finally, agricultural benefits (changes in farm income and consumer welfare) predicted to result in a future year have been adjusted to 1997 dollars from 2010 using a GDP price deflator. In this analysis, 2010 benefits were adjusted to 1997 dollars by multiplying by 0.6735, the ratio of the 1997 GDP price deflator (of 112.3 from:Council of Economic Advisers, 1997, Table B-3) to a projected 2010 GDP price index (of 167.16) forecasted from the trend between 1997 and 2007, obtained from the USDA baseline projections (U.S. Department of Agriculture, 1988b, electronic file Tab01.wk1).

3.2.4 Aggregation of Monetized Benefits

The total monetized benefit associated with attaining a given set of pollution changes in a given location is just the sum of the non-overlapping benefits associated with these changes. In theory, the total health-related social value of the reduction in pollution concentrations is:

$$\sum_{i=1}^{N}\sum_{j=1}^{J} WTP_i\left(B_{i,j}\right),$$

where B_{ij} is the benefit related to the j^{th} health endpoint (i.e., the reduction in probability of having to endure the j^{th} health problem) conferred on the i^{th} individual by the reduction in pollution concentrations, and $WTP_i(B_{ij})$ is the i^{th} individual's WTP for that benefit.

However, the reduction in probability of each health problem for each individual is not known, nor do we know each individual's WTP for each possible benefit he or she might receive. Therefore, in practice, benefits analysis estimates the value of a *statistical* health problem avoided. The benefit in the k^{th} location associated with the j^{th} health endpoint is just the change in incidence of the j^{th} health endpoint in the k^{th} location, Δy_{jk}, times the value of an avoided occurrence of the jth health endpoint.

Assuming that WTP to avoid the risk of a health effect varies from one individual to another, there is a *distribution* of WTPs to avoid the risk of that health effect. This population distribution has a mean. It is this population mean of WTPs to avoid or reduce the risk of the j^{th} health effect, $MWTP_j$, that is the appropriate value in the benefit analysis.[19] The monetized benefit associated with the j^{th} health endpoint resulting from attainment of standard(s) in the k^{th} location, then, is:

$$benefit_{jk} = \Delta y_{jk} \cdot MWTP_j$$

and total monetized benefit in the k^{th} location (TMB_k) may be written as the sum of the monetized benefits associated with all non-overlapping endpoints:

$$TMB_k = \sum_{j=1}^{N} \Delta y_{jk} \cdot MWTP_j \ .$$

The location- and health endpoint-specific incidence change, Δy_{jk}, is modeled as the population response to the change in pollutant concentrations in the k^{th} location. The discussion below uses particulate matter as an example but is equally applicable to any other pollutant, such as ozone. Assuming a log-linear C-R function, the change in incidence of the j^{th} health endpoint in the k^{th} location corresponding to a change in PM, ΔPM_k, in the k^{th} location is:

[19]The population of interest has not been defined. In a location-specific analysis, the population of interest is the population in that location. The MWTP is ideally the mean of the WTPs of all individuals in the location. There is insufficient information, however, to estimate the MWTP for any risk reduction in any particular location. Instead, estimates of MWTP for each type of risk reduction will be taken to be estimates of the MWTP in the United States as a whole, and it will be assumed that $MWTP_i$, i=1, ..., N in each location is approximately the same as in the United States as a whole.

$$\Delta y_{jk} = y_{jk} \cdot \left(e^{b_{jk} \cdot \Delta PM_k} - 1 \right) ,$$

where y_{jk} is the baseline incidence of the j^{th} health endpoint in the k^{th} location and β_{jk} is the value of β_j, the coefficient of PM in the C-R relationship between PM and the j^{th} health endpoint, in the k^{th} location.

This approach assumes that there is a *distribution* of β_j's across the United States, that is, that the value of β_j in one location may not be the same as the value of β_j in another location. The value of β_j in the k^{th} location is denoted as β_{jk}.

The total PM-related monetized benefit for the k^{th} location can now be rewritten as:

$$TMB_k = \sum_{j=1}^{N} y_{jk} \cdot \left(e^{b_{jk} \cdot \Delta PM_k} - 1 \right) \cdot MWTP_j ,$$

The total monetized PM-related benefit to be estimated for a location is thus a function of 2N parameters: the coefficient of PM, β_{jk}, in the C-R function for the j^{th} health (or welfare) endpoint, for j=1, ..., N, specific to the k^{th} location, and the population mean WTP to reduce the risk of the j^{th} health endpoint, $MWTP_j$, j=1, ..., N.

The above model assumes that total monetized benefit is the sum of the monetized benefits from all non-overlapping endpoints. If two or more endpoints were overlapping, or if one was contained within the other (as, for example, hospital admissions for Chronic Obstructive Pulmonary Disease - COPD - is contained within hospital admissions for "all respiratory illnesses"), then adding the monetized benefits associated with those endpoints would result in double (or multiple) counting of monetized benefits. If some endpoints that are not contained within endpoints included in the analysis are omitted, then the aggregated monetized benefits will be less than the total monetized benefits.

The total monetized benefit (TMB) is the sum of the total monetized benefits achieved in each location:

$$TMB = \sum_{k=1}^{K} TMB_k$$

where TMB_k denotes the total monetized benefit achieved in the k^{th} location, and K is the number of locations.

Theoretically, the nation-wide analysis could use location-specific C-R functions to estimate location-specific benefits. Total monetized benefits (TMB), then, would just be the sum of these location-specific benefits:

$$TMB = \sum_{k=1}^{K} TMB_k = \sum_{k=1}^{K} \sum_{j=1}^{N} y_{jk} \cdot \left(e^{b_{jk} \cdot \Delta PM_k} - 1 \right) \cdot MWTP_j ,$$

There are many locations in the United States, however, and the individual location-specific values of β_j (the β_{jk}'s) are not known.[20] Since the national incidence of the j^{th} health endpoint attributed to PM, I_j, is a continuous function of the set of β_{jk}'s, that is, since:

$$I_j = \sum_{k=1}^{K} \Delta y_{jk} = \sum_{k=1}^{K} y_{jk} \cdot \left(e^{b_{jk} \cdot \Delta PM_k} - 1 \right) ,$$

is a continuous function of the set of β_{jk}'s, there is some value of β_j , which can be denoted β_j*, that, if applied in *all* locations, would yield the same result as the proper set of location-specific β_{jk}'s. This follows from the Intermediate Value Theorem. While β_j* will result in overestimates of incidence in some locations, it will result in underestimates in others. If β_j* is applied in all locations, however, the *total regional* change in incidence will be correct. That is,

$$I_j = \sum_{k=1}^{K} \Delta y_{jk} = \sum_{k=1}^{K} y_{jk} \cdot \left(e^{b_j^* \cdot \Delta PM_k} - 1 \right) ,$$

$$= \sum_{k=1}^{K} y_{jk} \cdot \left(e^{b_{jk} \cdot \Delta PM_k} - 1 \right) .$$

The total regional monetized PM-related benefit can now be rewritten as:

$$TMB_k = \sum_{j=1}^{N} \sum_{k=1}^{K} y_{jk} \cdot \left(e^{b_j^* \cdot \Delta PM_k} - 1 \right) \cdot MWTP_j .$$

The total regional monetized (PM-related) benefit is thus a function of 2N population means: the β* for the j^{th} health (or welfare) endpoint (β_j* , for j=1, ..., N) and the population mean WTP to reduce the risk of the j^{th} health endpoint ($MWTP_j$, j=1, ..., N).

The above formulation of the total monetized benefits associated with a given set of changes in PM across K locations is applied to ozone as well. The set of health and welfare endpoints may be different for ozone, but the calculation of benefits is the same, with $\Delta ozone_k$ substituted for ΔPM_k everywhere.

Both the endpoint-specific coefficients (the ÿ$_j$'s) and the endpoint-specific mean WTPs (the $MWTP_j$'s) are uncertain. One approach to estimating the total monetized benefit is to simply use the mean values of the endpoint-specific coefficients and mean WTPs in the above formula. We term this approach the "simple mean." Alternatively, we can characterize not only the mean total monetized benefit but the

[20]This may also be true of the y_{ij}'s. It may be desirable to apply the uncertainty analysis used for the β's to these population parameters as well. In the current discussion, however, it is assumed that the location-specific incidences are known and therefore have no uncertainty associated with them. It is also assumed that $MWTP_i$ is the same in all locations.

distribution of possible values of total monetized benefit, using a Monte Carlo approach. The Monte Carlo approach has three steps. First, in each of 5000 iterations, we randomly select a value from the distribution of (national) incidence change of the health or welfare effect. Second, we randomly select a value from the distribution of unit dollar values for that health or welfare effect. And third, we multiply the two values. The result is a distribution of (5000) monetized benefits associated with the given health or welfare effect. From this distribution, we present the mean as well as the 5^{th} and 95^{th} percentiles. We discuss the background of the Monte Carlo in the following sub-section.

3.3 CHARACTERIZATION OF UNCERTAINTY

In any complex analysis using estimated parameters and inputs from numerous different models, there are likely to be many sources of uncertainty. This analysis is no exception. There are many inputs that are used to derive the final estimate of benefits, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological estimates of C-R functions, estimates of values (both from WTP and cost-of-illness studies), population estimates, income estimates, and estimates of the future state of the world, i.e. regulations, technology, and human behavior. Each of these inputs may be uncertain, and depending on their location in the benefits analysis, may have a disproportionately large impact on final estimates of total benefits. For example, emissions estimates are used in the first stage of the analysis. As such, any uncertainty in emissions estimates will be propagated through the entire analysis. When compounded with uncertainty in later stages, small uncertainties in emissions can lead to much larger impacts on total benefits.

Exhibit 3-3 summarizes the wide variety of sources for uncertainty in this analysis. Some key sources of uncertainty in each stage of the benefits analysis are:

- gaps in scientific data and inquiry
- variability in estimated relationships, such as C-R functions, introduced through differences in study design and statistical modeling
- errors in measurement and projection for variables such as population growth rates
- errors due to misspecification of model structures, including the use of surrogate variables, such as using PM_{10} when $PM_{2.5}$ is not available, excluded variables, and simplification of complex functions
- biases due to omissions or other research limitations.

Our approach to characterizing model uncertainty in the estimate of total benefits is to present a primary estimate, based on the best available scientific literature and methods, and to provide estimates of the effects of uncertainty about key analytical assumptions. However, in some cases, it was not possible to quantify uncertainty. For example, many benefits categories, while known to exist, do not have enough information available to provide a quantified or monetized estimate. The uncertainty regarding these endpoints is such that we could determine neither a primary estimate nor a plausible range of values. To the extent possible, we address uncertainty by presenting alternative calculations, supplemental calculations sensitivity analyses, and probabilistic assessments. We discuss each approach in turn.

Exhibit 3-3 Key Sources of Uncertainty in the Benefit Analysis

1. Uncertainties Associated With Concentration-Response Functions
-The value of the ozone- or PM-coefficient in each C-R function. -Application of a single C-R function to pollutant changes and populations in all locations. -Similarity of future year C-R relationships to current C-R relationships. -Correct functional form of each C-R relationship. -Extrapolation of C-R relationships beyond the range of ozone or PM concentrations observed in the study.
2. Uncertainties Associated With Ozone and PM Concentrations
-Estimating future-year baseline and hourly ozone and daily PM concentrations. -Estimating the change in ozone and PM resulting from the control policy.
3. Uncertainties Associated with PM Mortality Risk
-No scientific literature supporting a direct biological mechanism for observed epidemiological evidence. -Direct causal agents within the complex mixture of PM responsible for reported health effects have not been identified. -The extent to which adverse health effects are associated with low level exposures that occur many times in the year versus peak exposures. -Possible confounding in the epidemiological studies of $PM_{2.5}$, effects with other factors (e.g., other air pollutants, weather, indoor/outdoor air, etc.). -The extent to which effects reported in the long-term studies are associated with historically higher levels of PM rather than the levels occurring during the period of study. -Reliability of the limited ambient $PM_{2.5}$ monitoring data in reflecting actual $PM_{2.5}$ exposures.
4. Uncertainties Associated With Possible Lagged Effects
-What portion of the PM-related long-term exposure mortality effects associated with changes in annual PM levels would occur in a single year, and what portion might occur in subsequent years.
5. Uncertainties Associated With Baseline Incidence Rates
-Some baseline incidence rates are not location-specific (e.g., those taken from studies) and may therefore not accurately represent the actual location-specific rates. -Current baseline incidence rates may not well approximate what baseline incidence rates will be in the year 2030. -Projected population and demographics -- used to derive incidences – may not well approximate future-year population and demographics.
6. Uncertainties Associated With Economic Valuation
-Unit dollar values associated with health and welfare endpoints are only estimates of mean WTP and therefore have uncertainty surrounding them. -Mean WTP (in constant dollars) for each type of risk reduction may differ from current estimates due to differences in income or other factors.
7. Uncertainties Associated With Aggregation of Monetized Benefits
-Health and welfare benefits estimates are limited to the available C-R functions. Thus, unquantified benefit categories will cause total benefits to be underestimated.

3.3.1 Alternative and Supplementary Calculations

The alternative calculations included in this analysis are based on relatively plausible alternatives to the assumptions used in deriving the primary benefit estimates. We do not attempt to assign probabilities to these alternative calculations, as we believe this would only add to the uncertainty of the analysis or present a false picture about the precision of the results[21]. Instead, the reader is invited to examine the impact of applying the different assumptions on the estimate of total benefits. While it is possible to combine all of the alternative calculations with a positive impact on benefits to form a "high" estimate or all of the alternative calculations with a negative impact on benefits to form a "low" estimate, we do not recommend this because the probability of all of these alternative assumptions occurring simultaneously is likely to be very low. Instead, the alternative calculations are intended to demonstrate the sensitivity of our benefits results to key parameters which may be uncertain. Exhibit 3-4 summarizes the alternative calculations included in this analysis.

Exhibit 3-4 also summarizes supplemental calculations prepared for this analysis. Supplemental calculations are intended to provide additional information about specific health effects, but are not suitable for inclusion in the primary or alternative estimates due to concerns about double-counting of benefits or the high degree of uncertainty about the estimates. Results from the supplemental calculations can be found in Appendix A.

Alternative Calculations

The Dockery et al. (1993) estimate of the relationship between PM exposure and premature mortality is a plausible alternative to that based on the Pope et al. (1995) However, the Dockery et al. study had a more limited geographic scope (and a smaller study population) than the Pope et al. study. The Dockery et al. study also covered a broader age category (25 and older compared to 30 and older in the Pope et al. study) and followed the cohort for a longer period (15 years compared to 8 years in the Pope et al. study). For these reasons, the Dockery et al. study is considered to be a plausible alternative estimate of the avoided premature mortality incidences.

The value of statistical life years alternative calculation recognizes that individuals who die from air pollution related causes tend to be older than the average age of individuals in the VSL studies used to develop the $5.9 million value. To employ the value of statistical life-year (VSLY) approach, we first estimated the age distribution of those lives projected to be saved by reducing air pollution. Based on life expectancy tables, we calculate the life-years saved from each statistical life saved within each age and gender cohort. To value these statistical life-years, we hypothesized a conceptual model which depicted the relationship between the value of life and the value of life-years. The average number of life-years saved across all age groups for which data were available is 14 for PM-related mortality. The average for PM, in particular, differs from the 35-year expected remaining lifespan derived from existing wage-risk studies. Using the same distribution of value of life estimates used above, we estimated a distribution for the value of a life-year and combined it with the total number of estimated life-years lost.

[21] Some recent benefit-cost analyses in Canada and Europe (Holland et al., 1999; Lang et al., 1995) have estimated ranges of benefits by assigning *ad hoc* probabilities to ranges of parameter values for different endpoints. Although this does generate a quantitative estimate of an uncertainty range, the estimated points on these distributions are themselves highly uncertain and very sensitive to the subjective judgements of the analyst. To avoid these subjective judgements, we choose to allow the reader to determine the weights they would assign to alternative estimates.

Reversals in chronic bronchitis incidences are defined as those cases where an individual reported having chronic bronchitis at the beginning of the study period but reported not having chronic bronchitis in follow-up interviews at a later point in the study period. Since, by definition, chronic diseases are long-lasting or permanent, if the disease goes away it is not chronic. In the primary analysis, these reversals are given a value of zero. As an alternative calculation, we estimate reversals and value each as a case of the mildest form of chronic bronchitis.

The alternative calculation for residential visibility is based on the McClelland et al. (1991) study of WTP for visibility changes in Chicago and Atlanta. The residential visibility estimates from the available literature have been determined by the SAB to be inadequate for use in a primary estimate in a benefit-cost analysis, because they have not undergone rigorous peer review (EPA-SAB-COUNCIL-ADV-00-002, 1999). However, residential visibility is likely to have some value and the McClelland et al. study is probably the best in estimating the likely magnitude of the benefits of residential visibility improvements.

Exhibit 3-4 Alternative and Supplemental Benefits Calculations for the Tier II 2030 Control Scenario

Alternative/Supplemental Calculations	Description
Alternative Calculations	
PM-related premature mortality based on Dockery et al. (1993)	The Dockery,et al. study provides an alternative estimate of the relationship between chronic PM exposure and mortality.
Value of avoided premature mortality incidences based on statistical life years.	Calculate the incremental number of life-years lost from exposure to changes in ambient PM and use the value of a statistical life year based on a $5.9 million value of a statistical life.
Reversals in chronic bronchitis treated as lowest severity cases	Instead of omitting those cases of chronic bronchitis that reverse after a period of time, they are treated as being cases with the lowest severity rating.
Value of visibility changes in Eastern U.S. residential areas	Value of visibility changes outside of Class I areas are estimated for the Eastern U.S. based on the reported values for Chicago and Atlanta derived from McClelland et al. (1991).
Household soiling damage	Value of decreases in expenditures on cleaning are estimated using values derived from Manuel et al. (1982).
Avoided costs of reducing nitrogen loadings in East coast estuaries	Estuarine benefits in 12 East coast estuaries from reduced atmospheric nitrogen deposition are approximated using the avoided costs of removing or preventing loadings from terrestrial sources.
Uncertainty bounds of aggregate benefit totals	5^{th} and 95^{th} percentile values of the distribution of total estimated benefits for ozone, PM, and ozone + PM.
Supplemental Calculations	
Short-term mortality	The Schwartz et al. (1996) study provides an estimate of the relationship between acute PM exposure and mortality.
Post-neonatal mortality	The Woodruff et al. (1997) study provides an estimate of the relationship between chronic exposure and infant mortality.
Ozone mortality	Ozone-related mortality benefits estimated using a pooled analysis based on four U.S. studies.
Asthma Attacks	Due to the potential for overlap with health effects covered in the pooled estimate of MRADs and Any-of-19 Respiratory Symptoms, cases of PM-related moderate or worse asthma (Ostro et al. (1991)) and cases of both PM- and Ozone-related asthma attacks (Whittemore and Korn (1980)) are presented separately.
Restricted activity days	Restricted activity days are presented separately because they overlap with work loss days and minor restricted activity days.
Ozone-related cardiovascular disease	Burnett et al. (1997) provides an estimate of cardiovascular-related hospital admissions.

The alternative calculation for household soiling is based on the Manuel et al. (1982) study of consumer expenditures on cleaning and household maintenance. However, the data used to estimate household soiling damages in the Manuel et al. study is from a 1972 consumer expenditure survey and as such may not accurately represent consumer preferences in the future.

The alternative calculation for the avoided costs of reductions in nitrogen loadings is constructed by examining the avoided costs to surrounding communities of reduced nitrogen loadings for three case study estuaries (Albemarle-Pamlico Sounds, Chesapeake Bay, and Tampa Bay). The three case study estuaries are chosen because they have agreed upon nitrogen reduction goals and the necessary nitrogen control cost data. The estimated costs for these three case-study estuaries are then averaged and applied to nine other estuaries, chosen for their prominence in the eastern U.S.

Uncertainty bounds are provided as an alternative calculation for aggregate totals of benefits. The 5^{th} and 95^{th} percentile alternative calculations are estimated by holding air quality changes, population estimates, and other factors constant and determining the distribution of total benefits that would be generated by a large number of random draws from the distributions of C-R functions and economic valuation functions. These alternative calculations thus show how the primary estimate of benefits changes in response to uncertainty in the measurement of C-R and valuation functions.

Supplemental Calculations

Studies examining the relationship between short-term exposures and premature mortality can reveal what proportion of premature mortality is due to immediate response to daily variations in PM. There is only one short-term study (presenting results from 6 separate U.S. cities) that uses $PM_{2.5}$ as the metric of PM (Schwartz et al. (1996)). As such, the supplemental estimate for premature mortality related to short-term PM exposures is based on the pooled city-specific, short-term $PM_{2.5}$ results from Schwartz et al.

The estimated effect of PM exposure on premature mortality in infants (post-neonatal) is based on a single U.S. study (Woodruff et al. (1997)) that, on recommendation of the SAB, was deemed too uncertain to include in the primary analysis. Adding this endpoint to the primary benefits estimate would result in an increase in total benefits.

In previous regulatory analyses, estimated incidences of ozone-related premature mortality have been estimated as a primary endpoint. Based on recent advice from the Science Advisory Board (SAB) (EPA-SAB-Council-ADV-99-012, 1999), however, we have converted this endpoint to a supplemental estimate to avoid potential double-counting of benefits captured by the Pope et al. PM premature mortality endpoint. There are many studies of the relationship between ambient ozone levels and daily mortality levels. The supplemental estimate is calculated using results from only four U.S. studies (Ito and Thurston (1996), Kinney et al. (1995), Moolgavkar et al. (1995), and Samet et al. (1997)), based on the assumption that demographic and environmental conditions on average would be more similar between these studies and the conditions prevailing when this regulation is implemented.

Due to the potential for overlap with health effects covered in the pooled estimate of MRADs and Any-of-19 Respiratory Symptoms, cases of PM-related moderate or worse asthma (Ostro et al. (1991)) and cases of both PM- and ozone-related asthma attacks (Whittemore and Korn (1980)) are presented separately as supplemental calculations. To include them would lead to a potential double-counting of benefits related to the avoidance of asthma-related health effects.

Restricted activity days (Ostro, 1987) is another health effect that overlaps with endpoints included in the primary analysis. Restricted activity days are defined as work loss days, missed school days, days spent in bed, and other restricted activity days (Adams and Benson, 1992, p. 4). Health effects included in this definition overlap with health effects included in both measures of work loss days and minor restricted

activity days. To include both of these endpoints along with restricted activity days would lead to a double-counting of benefits, therefore restricted activity days are presented as a supplemental calculation.

The last supplemental calculation is an alternative measure of ozone-related cardiovascular disease. There are only two studies that are relevant for this endpoint, Burnett et al. (1997) and Burnett et al. (1999). Burnett et al. (1997) gives implausibly large estimates of cardiovascular disease. The link between ozone and cardiovascular problems is not as well established as that between ozone and respiratory problems. Other studies have not found a link between ozone and cardiovascular problems, and instead have found associations with other pollutants, like PM. Acknowledging the uncertainty in our estimate, we use only the results of the Burnett et al. (1999) study that focused on a narrow subset of cardiovascular problems, the relationship between ozone and abnormal heart rhythms or "dysrhythmias."

3.3.2 Sensitivity Analyses

In addition to alternative calculations and supplementary calculations, we will perform sensitivity analyses, briefly described in Exhibit 3-5. Sensitivity analyses, as opposed to alternative calculations, examine the sensitivity of estimated benefits results to less plausible alternatives to the assumptions used in the primary analysis. Sensitivity calculations also demonstrate the sensitivity of our benefits results to key analytical parameters. The sensitivity analyses calculated for this analysis will include the impact of a threshold assumption on Pope et al. (1995) mortality, alternative lag structures when valuing mortality, and the extrapolation of benefits from reduced nitrogen loadings to all East coast nutrient-sensitive estuaries. Results from the sensitivity analyses are presented in Appendix A.

Exhibit 3-5 Sensitivity Analyses for the Tier II 2030 Control Scenario

Sensitivity Analysis	Description
Threshold assumptions	Calculate the impact varying threshold assumptions have on the estimation of mortality incidence based on the Pope et al. (1995) study.
Alternative mortality lag structures	Calculate the impact different lag structures have on the estimation of benefits associated with avoided mortality incidence.
Avoided costs of reducing nitrogen loadings in East coast estuaries	Estuarine benefits attributed to 12 nutrient-sensitive East coast estuaries extrapolated to represent benefits associated with reductions in nitrogen at all nutrient-sensitive East coast estuaries.

3.3.3 Statistical Uncertainty Bounds

Although there are several sources of uncertainty affecting estimates of endpoint-specific benefits, the sources of uncertainty that are most readily quantifiable in this analysis are the C-R relationships and uncertainty about unit dollar values. The total dollar benefit associated with a given endpoint depends on how much the endpoint will change due to the final standard (e.g., how many premature deaths will be avoided) and how much each unit of change is worth (e.g., how much a premature death avoided is

worth).[22] Based on these distributions, we provide estimates of the 5th and 95th percentile values of the distribution of estimated benefits. However, we hasten to add that this omits important sources of uncertainty, such as the contribution of air quality changes, baseline population incidences, projected populations exposed, transferability of the C-R function to diverse locations, and uncertainty about premature mortality. Thus, a confidence interval based on the standard error would provide a misleading picture about the overall uncertainty in the estimates. The empirical evidence about uncertainty is presented where it is available.

Both the uncertainty about the incidence changes and uncertainty about unit dollar values can be characterized by *distributions*. Each "uncertainty distribution" characterizes our beliefs about what the true value of an unknown (e.g., the true change in incidence of a given health effect) is likely to be, based on the available information from relevant studies.[23] Unlike a sampling distribution (which describes the possible values that an *estimator* of an unknown value might take on), this uncertainty distribution describes our beliefs about what values the unknown value itself might be. Such uncertainty distributions can be constructed for each underlying unknown (such as a particular pollutant coefficient for a particular location) or for a function of several underlying unknowns (such as the total dollar benefit of a regulation). In either case, an uncertainty distribution is a characterization of our beliefs about what the unknown (or the function of unknowns) is likely to be, based on all the available relevant information. Uncertainty statements based on such distributions are typically expressed as 90 percent credible intervals. This is the interval from the fifth percentile point of the uncertainty distribution to the ninety-fifth percentile point. The 90 percent credible interval is a "credible range" within which, according to the available information (embodied in the uncertainty distribution of possible values), we believe the true value to lie with 90 percent probability.

The uncertainty about the total dollar benefit associated with any single endpoint combines the uncertainties from these two sources, and is estimated with a Monte Carlo method. In each iteration of the Monte Carlo procedure, a value is randomly drawn from the incidence distribution and a value is randomly drawn from the unit dollar value distribution, and the total dollar benefit for that iteration is the product of the two.[24] If this is repeated for many (e.g., thousands of) iterations, the distribution of total dollar benefits associated with the endpoint is generated.

Using this Monte Carlo procedure, a distribution of dollar benefits may be generated for each endpoint. The mean and median of this Monte Carlo-generated distribution are good candidates for a point estimate of total monetary benefits for the endpoint. As the number of Monte Carlo draws gets larger and larger, the Monte Carlo-generated distribution becomes a better and better approximation to the underlying uncertainty distribution of total monetary benefits for the endpoint. In the limit, it is identical to the underlying distribution.

---

[22] Because this is a regional analysis in which, for each endpoint, a single C-R function is applied everywhere, there are two sources of uncertainty about incidence: (1) statistical uncertainty (due to sampling error) about the true value of the pollutant coefficient in the location where the C-R function was estimated, and (2) uncertainty about how well any given pollutant coefficient approximates β^*.

[23] Although such an "uncertainty distribution" is not formally a Bayesian posterior distribution, it is very similar in concept and function (see, for example, the discussion of the Bayesian approach in Kennedy1990, pp. 168-172).

[24] This method assumes that the incidence change and the unit dollar value for an endpoint are stochastically independent.

3.3.4 Unquantified Benefits

In considering the monetized benefits estimates, the reader should remain aware of the limitations. One significant limitation of both the health and welfare benefits analyses is the inability to quantify many of the PM and ozone-induced adverse effects. For many health and welfare effects, such as PM-related materials damage, reliable C-R functions and/or valuation functions are not currently available. In general, if it were possible to monetize these benefits categories, the benefits estimates presented in this RIA would increase. In addition to unquantified benefits, there may also be environmental costs that we are unable to quantify. Several of these environmental cost categories are related to nitrogen deposition, while one category is related to the issue of ultraviolet light. The net effect of excluding benefit and disbenefit categories from the estimate of total benefits depends on the relative magnitude of the effects.

4 HEALTH BENEFITS

The most significant monetized benefits of reducing ambient concentrations of PM and ozone are attributable to reductions in health risks associated with air pollution. This Chapter describes individual effects and the methods used to quantify and monetize changes in the expected number of incidences of various health effects.

We estimate the incidence of adverse health effects using C-R functions based on PM and ozone. The changes in incidence of PM-related and ozone-related adverse health effects and corresponding monetized benefits associated with these changes are estimated separately. The PM- and ozone-related health endpoints for which C-R functions are estimated are shown in Exhibits 4-1 and 4-2, respectively. The unit monetary values for each of these endpoints, and associated uncertainty distributions, are presented in Exhibit 4-3. In some cases there are alternative and/or supplemental endpoints, studies, or unit dollar values that could be used in calculating the benefits of a change in pollution. These alternatives are presented where appropriate in Exhibits 4-1, 4-2, and 4-3 in italics to indicate that they are not used in the primary analysis but may be used in alternative analyses or used to supplement the existing analyses. Appendices B and C present the functional forms for each C-R function and how they were derived.

Issues relating to the calculation of changes in incidence and the monetization of these changes are discussed below for each endpoint. For some of the endpoint-pollutant combinations, there are several epidemiological studies that have estimated C-R functions. In these cases, the information in the multiple studies is pooled, so that the estimation of the change in incidence and the corresponding monetized value of that change is based on a synthesis of the information in all the available studies. A general discussion of pooling issues is provided above. A detailed description of the method used to pool multiple studies in this analysis is given below for those endpoints for which pooling was used.

Exhibit 4-1 PM-Related Health Endpoints

Endpoint	Population to Which Applied	PM Indicator	Study
Mortality			
Associated with long-term exposure	Ages 30+	$PM_{2.5}$	Pope et al. (1995)
Associated with long-term exposure [a]	*All ages*	*$PM_{2.5}$*	*Dockery et al. (1993)*
Chronic Illness			
Chronic Bronchitis	varies by study	varies by study	Multiple studies [b]
Hospital Admissions			
Respiratory	varies by study	varies by study	Multiple studies [b]
Cardiovascular	varies by study	varies by study	Multiple studies [b]
Asthma-related ER visits	< 65	PM_{10}	Schwartz et al. (1993)
Respiratory Symptoms/Illnesses Not Requiring Hospitalization			
Acute bronchitis	Ages 8-12	$PM_{2.5}$	Dockery et al. (1989)
Lower respiratory symptoms (LRS)	Ages 7-14	$PM_{2.5}$	Schwartz et al. (1994)
Upper respiratory symptoms (URS)	Asthmatics, ages 9-11	PM_{10}	Pope et al. (1991)
Shortness of breath (days with)	African-American asthmatics, ages 7-12	PM_{10}	Ostro et al. (1995)
Minor restricted activity day (MRAD)/ Any of 19 respiratory symptoms [c]	Ages 18-65	varies by study	Ostro and Rothschild (1989b), Krupnick et al. (1990)
Work loss days (WLDs)	Ages 18-65	$PM_{2.5}$	Ostro (1987)
Asthma	*Asthmatics, all ages*	*$PM_{2.5}$, PM_{10}*	*Ostro et al. (1991), Whittemore and Korn (1980)*
Restricted Activity Days (RADs)	*Ages 18-65*	*$PM_{2.5}$*	*Ostro (1987)*

[a] Italicized entries are either alternative or supplemental calculations to the endpoints and/or studies used in the primary analysis.

[b] The incidence changes, and the associated monetized benefits, predicted by several studies are pooled. The separate studies and the method of pooling are described below.

[c] The incidence changes, and the associated monetized benefits, from these two related endpoints are pooled.

Exhibit 4-2 Ozone-Related Health Endpoints

Endpoint	Population to Which Applied	Study
Chronic Illness		
Chronic asthma	non-asthmatic males, age 27+	McDonnell et al. (1999)
Hospital Admissions		
Respiratory	varies by study	Multiple studies [a]
Cardiovascular	varies by study	Multiple studies [a]
Asthma-related ER visits	varies by study	Multiple studies [a]
Symptoms/Illnesses Not Requiring Hospitalization		
Minor restricted activity day (MRAD) / Any of 19 respiratory symptoms [b]	Ages 18-65	Ostro and Rothschild (1989b), Krupnick et al. (1990)
Worker productivity	Working population	Crocker and Horst (1981) and EPA (1994)
Asthma attacks [c]	*Asthmatics, all ages*	*Whittemore and Korn (1980)*

[a] The incidence changes, and the associated monetized benefits, predicted by several studies are pooled. The separate studies and the method of pooling are described below.

[b] The incidence changes, and the associated monetized benefits, from these two related endpoints are pooled.

[c] Italicized entries are alternative or supplemental calculations to the endpoints and/or studies used in the primary analysis.

Exhibit 4-3 Unit Values for Economic Valuation of Health Endpoints (1997 $)

Health Endpoint	Mean Estimate [a]	Uncertainty Distribution [a]
Mortality		
Value of a statistical life	$5.9 million per statistical life	Weibull distribution, mean = $5.9 million; std. dev. = $3.98 million.
Value of a statistical life year [b]	*$2.8 million per statistical life (mean of 24 years of life saved)*	*Based on the Weibull distribution for the value of a statistical life, from which the value of a statistical life year is derived.*
Chronic Bronchitis		
WTP approach	$319,000 per case	A Monte Carlo-generated distribution, based on three underlying distributions.
Chronic Asthma		
	$31,000 per case	Triangular distribution centered at $31,000 over the interval [$23,000, $37,000].
Hospital Admissions		
Respiratory	— [c]	— [c]
Cardiovascular	— [c]	— [c]
Asthma-related ER visits	$279.55 per visit	Triangular distribution centered at $280 over the interval [$207.50, $387.63].
Respiratory Ailments Not Requiring Hospitalization		
Acute bronchitis	$55.26 per case	Continuous uniform distribution over [$15.96, $94.56].
Lower resp. Symptoms	$14.74 per symptom-day	Continuous uniform distribution over [$6.14, $23.33].
Upper resp. Symptoms	$23.33 per symptom-day	Continuous uniform distribution over [$8.60,$40.52].
Any of 19 acute respiratory symptoms/ minor restricted activity day (MRAD) [d]	Any of 19 symptoms: $22.10 per symptom-day MRAD: $46.66 per day	Any of 19 symptoms: Continuous uniform distribution over the interval [$0,$45.44]. MRAD: Triangular distribution centered at $46.66 over [$19.65, $74.91].
Shortness of breath	$6.51 per symptom-day	Continuous uniform distribution over [$0, $13.02]
Work loss days	$101.92 per day	None available
Worker productivity	Change in daily wages adjusted by regional variations in income	None available
Asthma - acute	*$39.30 per symptom-day*	*Continuous uniform distribution over [$14.74, $66.31]*
Asthma – moderate or worse	*$39.30*	*Continuous uniform distribution over [$14.74, $66.31]*
Restricted activity day (RAD)	*Based on MRAD valuation*	*Values based on MRAD valuation*

[a] The derivation of each of the estimates is discussed in the text. All WTP-based dollar values were obtained by multiplying rounded 1990 $ values used in the §812 Prospective Analysis by 1.228 to adjust to 1997 $. Entries in italics are not used in the primary benefits analysis.

[b] Based on a 5 percent discount rate, a value of $360,000 (rounded from $359,981) per life year (in 1997 $), a five-year lag structure, 1997 life expectancies, and 22,837 implied deaths (derived from the number of estimated life years lost). This is explained in greater detail in the text below.

[c] Definitions of endpoints vary by study. For example, "all respiratory illnesses" includes ICD-9 codes 460-519 in some studies, but only subsets of that group in other studies. Cost of illness unit dollar values were derived for each separate set of ICD codes for which a C-R model was estimated. These are given below.

[d] These two endpoints are pooled.

4.1 PREMATURE MORTALITY

Changes in PM concentrations on mortality may be estimated by a count of the expected number of deaths avoided due to a given reduction in PM concentrations. An alternative measure is to infer the number of years of life that are saved by a given reduction in PM concentrations: years of life that each individual was expected to live and that would have been lost had the reduction in PM concentrations not occurred. If life-years saved is used as a measure of the PM impact, then the value of a premature death avoided will likely depend on the age of the individual. Both measures of mortality are estimated in this analysis to provide a range of the possible cost of premature mortality.

Both ozone and particulate matter have been associated with increased risk of premature mortality, which is a very important health endpoint in this economic analysis due to the high monetary value associated with risks to life. There are two types of exposure to elevated levels of air pollution that may result in premature mortality. Acute (short-term) exposure (e.g., exposure on a given day) to peak pollutant concentrations may result in excess mortality on the same day or within a few days of the elevated exposure. Chronic (long-term) exposure (e.g., exposure over a period of a year or more) to levels of pollution that are generally higher may result in mortality in excess of what it would be if pollution levels were generally lower. The excess mortality that occurs will not necessarily be associated with any particular episode of elevated air pollution levels.

4.1.1 Short-Term Versus Long-Term Studies

There are two types of epidemiological studies that examine the relationship between mortality and exposure. Long-term studies (e.g., Pope et al., 1995) estimate the association between long-term (chronic) exposure to air pollution and the survival of members of a large study population over an extended period of time. Such studies examine the health endpoint of concern in relation to the general long-term level of the pollutant of concern, for example, relating annual mortality to some measure of annual pollutant level. Daily peak concentrations would impact the results only insofar as they affect the measure of long-term (e.g., annual) pollutant concentration. In contrast, short-term studies relate daily levels of the pollutant to daily mortality. By their basic design, daily studies can detect acute effects but cannot detect the effects of long-term exposures. A chronic exposure study design (a prospective cohort study, such as the Pope study) is best able to identify the long-term exposure effects, and may detect some of the short-term exposure effects as well. Because a long-term exposure study may detect some of the same short-term exposure effects detected by short-term studies, including both types of study in a benefit analysis would likely result in some degree of double counting of benefits. While the long-term study design is preferred, these types of studies are expensive to conduct and consequently there are relatively few well designed long-term studies.

4.1.2 Degree of Prematurity of Mortality

It is possible that the short-term studies are detecting an association between PM and mortality that is primarily occurring among terminally ill people. Critics of the use of short-term studies for policy analysis purposes correctly point out that an added risk factor that results in terminally ill people dying a few days or weeks earlier than they otherwise would have (referred to as “short-term harvesting”) is potentially included in the measured PM mortality “signal” detected in such a study. While some of the detected excess deaths may have resulted in a substantial loss of life (measuring loss of life in terms of lost years of remaining life), others may have lost a relatively short amount of lifespan. However, there is little

evidence to bear on this question. Studies by Spix et al (1993) and Pope et al. (1992) yield conflicting evidence, suggesting that harvesting may represent anywhere from zero to 50 percent of the deaths estimated in short-term studies. A recent study by Zeger et al. (1999), that focused exclusively on this issue, reported that short-term harvesting may be a quite small fraction of mortality.[25]

It is not likely, however, that the excess mortality reported in a long-term prospective cohort study like Pope et al. (1995) contains any significant amount of this short-term harvesting. The Cox proportional hazard statistical model used in the Pope study examines the question of survivability throughout the study period (ten years). Deaths that are premature by only a few days or weeks within the ten-year study period (for example, the deaths of terminally ill patients, triggered by a short duration PM episode) are likely to have little impact on the calculation of the average probability of surviving the entire ten-year interval.

4.1.3 Estimating PM-Related Premature Mortality

The benefits analysis estimates PM-related mortality using the $PM_{2.5}$ relationship from Pope et al. (1995). This decision reflects the Science Advisory Board's explicit recommendation for modeling the mortality effects of PM in both the completed §812 Retrospective Report to Congress and the ongoing §812 Prospective study (U.S. EPA, 1999b, p. 12). The Pope et al. study estimated the association between long-term (chronic) exposure to $PM_{2.5}$ and the survival of members of a large study population. This relationship is selected for use in the benefits analysis instead of short-term (daily pollution) studies for a number of reasons.

We selected the Pope et al. (1995) long-term study as providing the best available estimate of the relationship between PM and mortality. It is used alone– rather than considering the total effect to be the sum of estimated short-term and long-term effects– because summing creates the possibility of double-counting a portion of PM-related mortality. We selected the Pope et al. study in preference to other available long-term studies because it uses better statistical methods, has a much larger sample size, the longest exposure interval, and more locations (51 cities) in the United States, than other studies. It is unlikely that the Pope et al. study contains any significant amount of short-term harvesting. First, the health status of each individual tracked in the study is known at the beginning of the study period. Persons with known pre-existing serious illnesses were excluded from the study population. Second, the statistical model used in the Pope study examines the question of survivability throughout the study period (ten years). Deaths that are premature by only a few days or weeks within the ten-year study period (for example, the deaths of terminally ill patients, triggered by a short duration PM episode) are likely to have little impact on the calculation of the average probability of surviving the entire ten year interval. In relation to the "Six-cities" study by Dockery et al. (1993), the Pope et al. study found a smaller increase in excess mortality for a given PM air quality change.

It is currently unknown whether there is a time lag (a delay between changes in PM exposures and changes in mortality rates) in the chronic PM/premature mortality relationship. The existence of such a lag is important for the valuation of premature mortality incidences because economic theory suggests that benefits occurring in the future should be discounted. Although there is no specific scientific evidence of the existence or structure of a PM effects lag, current scientific literature on adverse health effects, such as

[25]Zeger et al. (1999, p. 171) reported that: "The TSP-mortality association in Philadelphia is inconsistent with the harvesting-only hypothesis, and the harvesting-resistant estimates of the TSP relative risk are actually larger – not smaller – than the ordinary estimates."

those associated with PM (e.g., smoking related disease) and the difference in the effect size between chronic exposure studies and daily mortality studies suggest that all incidences of premature mortality reduction associated with a given incremental change in PM exposure probably would not occur in the same year as the exposure reduction. This same smoking-related literature implies that lags of up to a few years are plausible. Following explicit advice from the SAB, we assume a five-year lag structure, with 25 percent of premature deaths occurring in the first year, another 25 percent in the second year, and 16.7 percent in each of the remaining three years (EPA-SAB-COUNCIL-ADV-00-001, 1999). Readers should note that the selection of a five-year lag structure is not directly supported by any PM-specific literature. Rather, it is intended to be a best guess at the appropriate distribution of avoided incidences of PM-related mortality.

Alternative Calculation: PM-Related Mortality Based on Dockery et al. (1993)

As an alternative to Pope et al. (1995), this analysis calculates the impact of PM on mortality using Dockery et al. (1993), another long-term PM-mortality study. Dockery et al. (1993) examined the relationship between PM exposure and mortality in a cohort of 8,111 individuals aged 25 and older, living in six U.S. cities. They surveyed these individuals in 1974-1977 and followed their health status until 1991. While they used a smaller sample of individuals from fewer cities than the study by Pope et al., they used improved exposure estimates, a slightly broader study population (adults aged 25 and older), and a follow-up period nearly twice as long as that of Pope et al. (1995). Perhaps because of these differences, Dockery et al. study found a larger effect of PM on premature mortality than that found by Pope et al.

Sensitivity Calculation: Mortality Lag Structure

To account for the uncertainty about when PM-related mortality will not occur in relation to the year that air pollution is reduced, we examine the sensitivity of mortality-related benefits to alternative lag structures. Exhibit 4-4 presents the lags that are used in these sensitivity calculations. As stated earlier, the primary analysis uses a five-year lag structure in the valuation of mortality and chronic bronchitis, with incidence apportioned as follows: 25 percent in the first year, 25 percent in the second year, and 16.67 percent in the last three years. To examine the effect alternate lag-structures have on the estimation of both mortality and chronic bronchitis valuation, the mortality benefits will be calculated using five alternative lag structures. Lag 1 will apportion the occurrence of all incidence to the first year. Valuation of these cases will not be discounted. In lag 2, based on the length of the study period for the Dockery et al. (1993) study, 100 percent of mortality incidence occurs in fifteen years from the modeled future-year. Lag 3, based on the length of the study period for the Pope et al. (1995) study, assigns 100 percent of the occurrence of mortality incidence to the eighth year out from the modeled future-year. Lag 4 front loads the occurrence of mortality incidence. Incidence is apportioned in decreasing amounts out to fifteen years. Lag 5 apportions incidence over fifteen years, assigning a lesser percentage of incidence in the beginning years, with the percentage of incidence increasing over time out to fifteen years. The latter two lag structures are intended to show how the distribution of incidences within a lag period affects benefit estimates.

Exhibit 4-4 Mortality Lag Structure

Year	Primary	Sensitivity 1	Sensitivity 2	Sensitivity 3	Sensitivity 4	Sensitivity 5
1	25	100	0	0	30	1
2	25	0	0	0	25	1
3	16.67	0	0	0	15	1
4	16.67	0	0	0	6	2
5	16.67	0	0	0	4	2
6	0	0	0	0	3	2
7	0	0	0	0	3	2
8	0	0	0	100	3	3
9	0	0	0	0	2	3
10	0	0	0	0	2	3
11	0	0	0	0	2	4
12	0	0	0	0	2	6
13	0	0	0	0	1	15
14	0	0	0	0	1	25
15	0	0	100	0	1	30

Sensitivity Calculation: Ozone-Related Mortality

Epidemiological studies suggest that there may be a link between ozone exposures and premature mortality, however possible confounding with PM-related mortality precludes its inclusion in the primary analysis. As an alternative, an ozone-related mortality meta-analysis was conducted to provide an alternative calculation of mortality incidence. Using a random-effects pooling procedure, we take the incidence estimates of four U.S. ozone-related mortality studies -- Ito and Thurston (1996), Kinney et al. (1995), Moolgavkar et al. (1995), and Samet et al. (1997) -- and estimate the mortality incidence changes for a given rule. For a complete discussion of ozone mortality and the pooling procedure, see the TSD for the proposed Tier II rule (Abt Associates, 1999).

4.1.4 Valuing Premature Mortality

Two methods for valuing avoided premature mortality are presented in this analysis. The first is the "statistical lives lost" approach. Using this approach, the value of a statistical death is estimated to be $5.9 million (in 1997 $). The second valuation approach is the "statistical life years lost" approach. Using this approach, the value of an avoid premature death depends on the age at which the individual dies. The average value for an avoided PM-related premature death, however, is $2.8 million (in 1997 $) (representing an average of 24 years of life saved, based on 1997 life expectancy estimates). In each case, we assume for this analysis that some of the incidences of premature mortality related to PM exposures occur in a distributed fashion over the five years following exposure (the five-year mortality lag). To take

this into account in the valuation of reductions in premature mortalities, we apply an annual five percent discount rate to the value of premature mortalities occurring in future years.[26]

Statistical Lives Lost

The "statistical lives lost" value of $5.9 million represents an intermediate value from a variety of estimates that appear in the economics literature, and is a value that EPA has frequently used in RIAs for other rules. This estimate is the mean of a distribution fitted to the estimates from 26 value-of-life studies identified in the §812 study as "applicable to policy analysis." The approach and set of selected studies mirrors that of Viscusi (1992) (with the addition of two studies), and uses the same criteria used by Viscusi in his review of value-of-life studies. The $5.9 million estimate is consistent with Viscusi's conclusion (updated to 1997 $) that "most of the reasonable estimates of the value of life are clustered in the $3.7 to $8.6 million range." Uncertainty associated with the valuation of premature mortality is expressed through a Weibull distribution with a standard deviation of $3.98 million (IEc 1992, p. 2).

Five of the 26 studies are contingent valuation (CV) studies, which directly solicit WTP information from subjects; the rest are wage-risk studies, which base WTP estimates on estimates of the additional compensation demanded in the labor market for riskier jobs. The 26 studies are listed in Exhibit 4-5. The references for all but Gegax et al. (1985) and V.K. Smith (1983) may be found in Viscusi (1992). Although each of the studies estimated the mean WTP (MWTP) for a given reduction in mortality risk, the amounts of reduction in risk being valued were not necessarily the same across studies, nor were they necessarily the same as the amounts of reduction in mortality risk that would actually be conferred by a given reduction in ambient concentrations. The transferability of estimates of the value of a statistical life from the 26 studies to this analysis rests on the assumption that, within a reasonable range, WTP for reductions in mortality risk is linear in risk reduction, or equivalently, that the marginal willingness to pay curve is horizontal within a reasonable range. For example, suppose a study estimates that the average WTP for a reduction in mortality risk of 1/100,000 is $30. Suppose, however, that the actual mortality risk reduction resulting from a given air quality improvement is 1/10,000. If WTP for reductions in mortality risk is linear in risk reduction, then a WTP of $30 for a reduction of 1/100,000 implies a WTP of $300 for a risk reduction of 1/10,000 (which is ten times the risk reduction valued in the study). Under the assumption of linearity, the estimate of the value of a statistical life does not depend on the particular amount of risk reduction being valued.

[26]The choice of a five percent discount rate is based on the technical recommendation of the SAB for computing the value of a statistical life-year (EPA-SAB-COUNCIL-ADV-00-002, 1999).

Exhibit 4-5 Summary of Mortality Valuation Estimates

Study	Type of Estimate	Valuation (millions 1997 $)
Kneisner and Leeth (1991) (US)	Labor Market	0.7
Smith and Gilbert (1984)	Labor Market	0.9
Dillingham (1985)	Labor Market	1.1
Butler (1983)	Labor Market	1.4
Miller and Guria (1991)	Contingent Valuation	1.5
Moore and Viscusi (1988)	Labor Market	3.1
Viscusi et al. (1991)	Contingent Valuation	3.3
Gegax et al. (1985; 1991)	Contingent Valuation	4.1
Marin and Psacharopoulos (1982)	Labor Market	3.4
Kneisner and Leeth (1991) (Australia)	Labor Market	4.1
Gerking et al. (1988)	Contingent Valuation	4.2
Cousineau et al. (1988; 1992)	Labor Market	4.4
Jones-Lee (1989)	Contingent Valuation	4.7
Dillingham (1985)	Labor Market	4.9
Viscusi (1978; 1979)	Labor Market	5.0
R.S. Smith (1976)	Labor Market	5.6
V.K. Smith (1983)	Labor Market	5.8
Olson (1981)	Labor Market	6.4
Viscusi (1981)	Labor Market	8.0
R.S. Smith (1974)	Labor Market	8.8
Moore and Viscusi (1988)	Labor Market	9.0
Kneisner and Leeth (1991) (Japan)	Labor Market	9.3
Herzog and Schlottman (1987; 1990)	Labor Market	11.2
Leigh and Folson (1984)	Labor Market	11.9
Leigh (1987)	Labor Market	12.8
Garen (1988)	Labor Market	16.6

Source: Viscusi (1992, Table 4.1).

Although the particular amount of mortality risk reduction being valued in a study may not affect the transferability of the WTP estimate from the study to this analysis, the characteristics of the study subjects and the nature of the mortality risk being valued in the study could be important. Certain characteristics of both the population affected and the mortality risk facing that population are believed to affect the MWTP to reduce the risk. The appropriateness of the MWTP estimates from the 26 studies for valuing the mortality-related benefits of reductions in ambient air concentrations therefore depends not only on the quality of the studies (i.e., how well they measure what they are trying to measure), but also on (1) the extent to which the subjects in the studies are similar to the population affected by changes in ambient air concentrations and (2) the extent to which the risks being valued are similar.

Focusing on the wage-risk studies, which make up the substantial majority of the 26 studies relied upon, the likely differences between (1) the subjects in these studies and the population affected by changes in air concentrations and (2) the nature of the mortality risks being valued in these studies and the nature of air pollution-related mortality risk are considered. The direction of bias in which each difference is likely to result is also considered.

Compared with the subjects in wage-risk studies, the population believed to be most affected by air pollution (i.e., the population that would receive the greatest mortality risk reduction associated with a given reduction in air concentrations) is, on average, older and probably more risk averse. For example, citing Schwartz and Dockery (1992) and Ostro et al. (1996), Chestnut (1995) estimated that approximately 85 percent of those who die prematurely from ambient air pollution-related causes are over 65. The average age of subjects in wage-risk studies, in contrast, is well under 65.

There is also reason to believe that those over 65 are, in general, more risk averse than the general population while workers in wage-risk studies are likely to be less risk averse than the general population. Although Viscusi's (1992) list of recommended studies excludes studies that consider only much-higher-than-average occupational risks, there is nevertheless likely to be some selection bias in the remaining studies -- that is, these studies are likely to be based on samples of workers who are, on average, more risk-loving than the general population. In contrast, older people as a group exhibit more risk averse behavior.

In addition, it might be argued that because the elderly have greater average wealth than those younger, the affected population is also wealthier, on average, than wage-risk study subjects, who tend to be blue collar workers. It is possible, however, that among the elderly it is largely the poor elderly who are most vulnerable to air pollution-related mortality risk (e.g., because of generally poorer health care). If this is the case, the average wealth of those affected by a reduction in air concentrations relative to that of subjects in wage-risk studies is uncertain.

The direction of bias resulting from the age difference is unclear, particularly because age is confounded by risk aversion (relative to the general population). It could be argued that, because an older person has fewer expected years left to lose, his WTP to reduce mortality risk would be less than that of a younger person. This hypothesis is supported by one empirical study, Jones-Lee et al.(1985), that found the value of a statistical life at age 65 to be about 90 percent of what it is at age 40. Citing the evidence provided by Jones-Lee et al., Chestnut (1995) assumed that the value of a statistical life for those 65 and over is 75 percent of what it is for those under 65.

The greater risk aversion of older people, however, implies just the opposite. Citing Ehrlich and Chuma (1990), Industrial Economics Inc. (1992) noted that "older persons, who as a group tend to avoid health risks associated with drinking, smoking, and reckless driving, reveal a greater demand for reducing mortality risks and hence have a greater implicit value of a life year." That is, the more risk averse behavior of older individuals suggests a greater WTP to reduce mortality risk.

There is substantial evidence that the income elasticity of WTP for health risk reductions is positive (Alberini et al., 1997; Gerking et al., 1988; Jones-Lee et al., 1985; Loehman et al., 1982; Mitchell et al., 1986), although there is uncertainty about the exact value of this elasticity). Individuals with higher incomes (or greater wealth) should, then, be willing to pay more to reduce risk, all else equal, than individuals with lower incomes or wealth. Whether the average income or level of wealth of the population affected by ambient air pollution reductions is likely to be significantly different from that of subjects in wage-risk studies, however, is unclear.

Finally, although there may be several ways in which job-related mortality risks differ from air pollution-related mortality risks, the most important difference may be that job-related risks are incurred voluntarily whereas air pollution-related risks are incurred involuntarily.

There is some evidence that people will pay more to reduce involuntarily incurred risks than risks incurred voluntarily (e.g., Violette and Chestnut, 1983). Job-related risks are incurred voluntarily whereas air pollution-related risks are incurred involuntarily. If this is the case, WTP estimates based on wage-risk studies may be downward biased estimates of WTP to reduce involuntarily incurred ambient air pollution-related mortality risks.

The potential sources of bias in an estimate of MWTP to reduce the risk of air pollution related mortality based on wage-risk studies are summarized in Exhibit 4-6. Although most of the individual factors tend to have a downward bias, the overall effect of these biases is unclear.

Exhibit 4-6 Potential Sources of Bias in Estimates of Mean WTP to Reduce the Risk of PM Related Mortality Based on Wage-Risk Studies

Factor	Likely Direction of Bias in Mean WTP Estimate
Age	Uncertain
Degree of Risk Aversion	Downward
Income	Downward, if the elderly affected are a random sample of the elderly. It is unclear, if the elderly affected are the poor elderly.
Risk Perception: Voluntary vs. Involuntary risk	Downward

Alternative Calculation: Statistical Life-Years Lost

In an alternative calculation, we value statistical life-years, rather than valuing statistical lives. Moore and Viscusi (1988) value a statistical life-year lost, by assuming that the WTP to save a statistical life is the value of a single year of life times the expected number of years of life remaining for an individual. They suggest that a typical respondent in a mortal risk study has a life expectancy of an additional 35 years. Using a mean estimate of $4.8 million (1990 $) to save a statistical life, their approach yields an estimate of $137,000 per life-year lost or saved, assuming no discounting. If an individual discounts future additional years using a standard discounting procedure, the value of each life-year lost must be greater than the value assuming no discounting. Using a 35 year life expectancy, a $4.8 million value of a statistical life, and a five percent discount rate, the implied value of each life-year lost is $293,000 (1990 $). This is $360,000 in 1997 dollars.

This analysis assumes a value of a statistical life year lost of $360,000 and a five percent discount rate, consistent with the "statistical lives lost" value of $5.9 million. In addition, the "statistical lives lost" analysis must accommodate the five-year lag structure. For each person dying at a given age, using the expected number of years remaining for that age, based on 1997 life expectancy tables (National Center for Health Statistics, 1999, Table 5), and a VSLY of $360,000, we calculate the present discounted value (discounted back to the beginning of the year of death) for that person. All values are then discounted back to the beginning of 2030, whether the individual dies in 2030 or in a subsequent year. The present

discounted value (discounted back to the beginning of 2030) of an avoided premature mortality will vary from one individual to another, depending on the age of the individual at death and on the extent of lag between exposure and death. The age at death determines the expected number of life years lost, while the extent of lag between exposure and death determines the amount of discounting needed. The average value of an avoided incidence of PM-related premature mortality, however, is $2.8 million (in 1997 $), corresponding to 24 years of life.

4.2 CHRONIC ILLNESS

Onset of bronchitis and asthma, two chronic illnesses, have both been associated with exposure to air pollutants. Three studies have linked the onset of chronic bronchitis in adults to particulate matter; one study has linked the onset of chronic asthma in adults to ozone. These results are consistent with research that has found chronic exposure to pollutants leads to declining pulmonary functioning (Abbey et al., 1998; Ackermann-Liebrich et al., 1997; Detels et al., 1991).

4.2.1 Chronic Bronchitis

We estimate the changes in the new cases of chronic bronchitis using the studies by Schwartz (1993), Abbey et al. (1993), and Abbey et al. (1995b). The Schwartz study is somewhat older and uses a cross-sectional design, however, it is based on a national sample, unlike the Abbey et al. studies which are based on a sample of California residents. We first pool the estimates from the two studies by Abbey et al. – since they are based on the same sample population and simply use different measures of PM – and then pool this estimate with that from Schwartz.

Three studies that have estimated C-R functions for PM and chronic bronchitis were pooled in this analysis. These studies are listed in Exhibit 4-7.

Exhibit 4-7 Chronic Bronchitis Studies

Location	Study	Pollutants Used in Final Model	Age of Study Population
California	Abbey et al. (1993)	PM_{10}	>26
California	Abbey et al. (1995b)	$PM_{2.5}$	>26
United States	Schwartz (1993)	PM_{10}	>29

Schwartz (1993) examined survey data collected from 3,874 adults ranging in age from 30 to 74, and living in 53 urban areas in the U.S. The survey was conducted between 1971 and 1975, as part of the National Health and Nutrition Examination Survey, and is representative of the non-institutionalized U.S. population. Schwartz (1993, Table 3) reported chronic bronchitis prevalence rates in the study population by age, race, and gender. Non-white males under 52 years old had the lowest rate (1.7%) and white males 52 years and older had the highest rate (9.3%). The study examined the relationship between the prevalence of reported chronic bronchitis and annual levels of total suspended particulates (TSP), collected in the year prior to the survey.

Abbey et al. (1993) surveyed 3,914 adult Seventh Day Adventists living in California, and estimated the relationship between annual mean ambient TSP, ozone and SO_2 and the onset of certain chronic respiratory symptoms (including airway obstructive disease (AOD), chronic bronchitis, and asthma) that were not present at the beginning of the study. The initial survey was conducted in 1977 and the final survey in 1987. To ensure a better estimate of exposure, the study participants had to have been living in the same area for an extended period of time. TSP was significantly linked to new cases of AOD and chronic bronchitis, but not to asthma or the severity of asthma. Ozone was not linked to the incidence of new cases of any endpoint, but ozone was linked to the severity of asthma. No effect was found for SO_2.

A later study by Abbey et al. (1995b) examined the relationship between estimated $PM_{2.5}$ (annual mean from 1966 to 1977), PM_{10} (annual mean from 1973 to 1977) and TSP (annual mean from 1973 to 1977) and the same chronic respiratory symptoms in a sample population of 1,868 Californian Seventh-Day Adventists. The initial survey was conducted in 1977 and the final survey in 1987. To ensure a better estimate of exposure, the study participants had to have been living in the same area for an extended period of time. In single-pollutant models, there was a statistically significant $PM_{2.5}$ relationship with development of chronic bronchitis, but not for AOD or asthma; PM_{10} was significantly associated with chronic bronchitis and AOD; and TSP was significantly associated with all cases of all three chronic symptoms. Other pollutants were not examined.

Alternative Calculation: Chronic Bronchitis Reversals

In developing the C-R functions for chronic bronchitis, it is necessary to estimate its annual incidence rate. The annual incidence rate is derived by taking the number of new cases (234), dividing by the number of individuals in the sample (3,310), as reported by Abbey et al.(1993, Table 3), dividing by the ten years covered in the sample, and then multiplying by one minus the reversal rate (the percentage of reversals is estimated to be 46.6% based on Abbey et al. (1995a, Table 1)). Reversals refer to those cases of chronic bronchitis that were reported at the start of the Abbey et al. survey, but were subsequently not reported at the end of the survey. Since we assume that chronic bronchitis is a permanent condition, we subtract these reversals. Nevertheless, reversals may likely represent a real effect that should be included in the analysis. To allow for this possibility, we present an estimate of reversals in an alternative calculation in which reversals are considered to be chronic bronchitis cases of the lowest severity level, as described below.

Valuing Chronic Bronchitis

PM-related chronic bronchitis is expected to last from the initial onset of the illness throughout the rest of the individual's life. WTP to avoid chronic bronchitis would therefore be expected to incorporate the present discounted value of a potentially long stream of costs (e.g., medical expenditures and lost earnings) and pain and suffering associated with the illness. Two studies, Viscusi et al. (1991) and Krupnick and Cropper (1992), provide estimates of WTP to avoid a case of chronic bronchitis.

The Viscusi et al. (1991) and the Krupnick and Cropper (1992) studies were experimental studies intended to examine new methodologies for eliciting values for morbidity endpoints. Although these studies were not specifically designed for policy analysis, we believe the studies provide reasonable estimates of the WTP for chronic bronchitis. As with other contingent valuation studies, the reliability of the WTP estimates depends on the methods used to obtain the WTP values. The Viscusi et al. and the Krupnick and

Cropper studies are broadly consistent with current contingent valuation practices, although specific attributes of the studies may not be.

The study by Viscusi et al. uses a sample that is larger and more representative of the general population than the study by Krupnick and Cropper (which selects people who have a relative with the disease). Thus, the valuation for the high-end estimate is based on the distribution of WTP responses from Viscusi et al. The WTP to avoid a case of pollution-related chronic bronchitis (CB) is derived by starting with the WTP to avoid a severe case of chronic bronchitis, as described by Viscusi et al. (1991), and adjusting it downward to reflect (1) the decrease in severity of a case of pollution-related CB relative to the severe case described in the Viscusi et al. study, and (2) the elasticity of WTP with respect to severity reported in the Krupnick and Cropper study. Because elasticity is a marginal concept and because it is a function of severity (as estimated from Krupnick et al., 1992), WTP adjustments were made incrementally, in one percent steps. A severe case of CB was assigned a severity level of 13 (following Krupnick and Cropper). The WTP for a one percent decrease in severity is given by:

$$WTP_{0.99\,sev} = WTP_{sev} \cdot (1 - 0.01 \cdot e) \ ,$$

where sev is the original severity level (which, at the start, is 13) and e is the elasticity of WTP with respect to severity. Based on the regression in Krupnick and Cropper (1992) (see below), the estimate of e is 0.18*sev. At the mean value of sev (6.47), e = 1.16. As severity decreases, however, the elasticity decreases. Using the regression coefficient of 0.18, the above equation can be rewritten as:

$$WTP_{0.99\,sev} = WTP_{sev} \cdot (1 - 0.01 \cdot 0.18 sev) \ .$$

For a given WTP_{sev} and a given coefficient of sev (0.18), the WTP for a 50 percent reduction in severity can be obtained iteratively, starting with sev =13, as follows:

$$WTP_{12.87} = WTP_{0.99 \cdot 13} = WTP_{13} \cdot (1 - 0.01 \cdot 0.18 \cdot 13)$$

$$WTP_{12.74} = WTP_{0.99 \cdot 12.87} = WTP_{12.87} \cdot (1 - 0.01 \cdot 0.18 \cdot 12.87)$$

$$WTP_{12.61} = WTP_{0.99 \cdot 12.74} = WTP_{12.74} \cdot (1 - 0.01 \cdot 0.18 \cdot 12.74)$$

and so forth. This iterative procedure eventually yields $WTP_{6.5}$, or WTP to avoid a case of chronic bronchitis that is of "average" severity.

The derivation of the WTP to avoid a case of pollution-related chronic bronchitis is based on three components, each of which is uncertain: (1) the WTP to avoid a case of severe CB, as described in the Viscusi et al. (1991) study, (2) the severity level of an average pollution-related case of CB (relative to that

of the case described by Viscusi et al.), and (3) the elasticity of WTP with respect to severity of the illness. Because of these three sources of uncertainty, the WTP is uncertain. Based on assumptions about the distributions of each of the three uncertain components, a distribution of WTP to avoid a pollution-related case of CB was derived by Monte Carlo methods. The mean of this distribution, which was about $319,000, is taken as the central tendency estimate of WTP to avoid a pollution-related case of CB. Each of the three underlying distributions is described briefly below.

1. The distribution of WTP to avoid a severe case of CB was based on the distribution of WTP responses in the Viscusi et al. (1991) study. Viscusi et al. derived respondents' implicit WTP to avoid a statistical case of chronic bronchitis from their WTP for a specified reduction in risk. The mean response implied a WTP of about $1,228,000 (1997 $)[27]; the median response implied a WTP of about $651,000 (1997 $). However, the extreme tails of distributions of WTP responses are usually considered unreliable. Because the mean is much more sensitive to extreme values, the median of WTP responses is often used rather than the mean. Viscusi et al. report not only the mean and median of their distribution of WTP responses, however, but the decile points as well. The distribution of reliable WTP responses from the Viscusi et al. study could therefore be approximated by a discrete uniform distribution giving a probability of 1/9 to each of the first nine decile points. This omits the first five and the last five percent of the responses (the extreme tails, considered unreliable). This trimmed distribution of WTP responses from the Viscusi et al. study was assumed to be the distribution of WTPs to avoid a severe case of CB. The mean of this distribution is about $884,000 (1997 $).

2. The distribution of the severity level of an average case of pollution-related CB was modeled as a triangular distribution centered at 6.5, with endpoints at 1.0 and 12.0. These severity levels are based on the severity levels used in Krupnick and Cropper (1992), which estimated the relationship between ln(WTP) and severity level, from which the elasticity is derived. The most severe case of CB in that study is assigned a severity level of 13. The mean of the triangular distribution is 6.5. This represents a 50 percent reduction in severity from a severe case.

3. The elasticity of WTP to avoid a case of CB with respect to the severity of that case of CB is a constant times the severity level. This constant was estimated by Krupnick and Cropper (1992) in the regression of ln(WTP) on severity, discussed above. This estimated constant (regression coefficient) is normally distributed with mean = 0.18 and standard deviation = 0.0669 (obtained from Krupnick and Cropper).

The distribution of WTP to avoid a case of pollution-related CB was generated by Monte Carlo methods, drawing from the three distributions described above. On each of 16,000 iterations (1) a value was selected from each distribution, and (2) a value for WTP was generated by the iterative procedure described above, in which the severity level was decreased by one percent on each iteration, and the corresponding WTP was derived. The mean of the resulting distribution of WTP to avoid a case of pollution-related CB was $319,000.

This WTP estimate is reasonably consistent with full COI estimates derived for chronic bronchitis, using average annual lost earnings and average annual medical expenditures reported by Cropper and Krupnick (1990) Using a 5 percent discount rate and assuming that (1) lost earnings continue until age 65, (2) medical expenditures are incurred until death, and (3) life expectancy is unchanged by chronic

[27]There is an indication in the Viscusi et al. (1991) paper that the dollar values in the paper are in 1987 dollars. Under this assumption, the dollar values were converted to 1997 dollars.

bronchitis, the present discounted value of the stream of medical expenditures and lost earnings associated with an average case of chronic bronchitis is estimated to be about $109,000 for a 30 year old, about $105,000 for a 40 year old, about $96,000 for a 50 year old, and about $55,000 for a 60 year old. A WTP estimate would be expected to be greater than a full COI estimate, reflecting the willingness to pay to avoid the pain and suffering associated with the illness. The WTP estimate of $319,000 is from 2.9 times the full COI estimate (for 30 year olds) to 5.8 times the full COI estimate (for 60 year olds). The midpoint of the COI estimates across the range of ages of $82,000 per case is used as an alternative valuation estimate for reduced incidence of chronic bronchitis.

Alternative Calculation: Valuing Chronic Bronchitis Reversals

In an alternative calculation, we estimate chronic bronchitis reversals and value them using the same method used to value cases of chronic bronchitis. However, instead of allowing the severity level to range from one to 13, we value all reversals at a severity level of one. This yields a WTP estimate of $140,000 for each chronic bronchitis reversal.

4.2.2 Chronic Asthma

In a number of studies ozone, PM, and even CO have been linked to acute asthmatic complaints (e.g., Ostro et al., 1995; Sheppard et al., 1999; Whittemore et al., 1980), however there is more limited evidence regarding the link between air pollution and the development of asthma. The best evidence points to ozone. Abbey et al. (1991; 1993) reported a significant link between ozone and the development of asthma, and Portney and Mullahy (1990) found ozone linked to sinusitis and hay fever. A review of research data by the EPA (1996a, p. 9-35) concluded that prolonged ozone exposure causes structural changes in several regions of the respiratory tract, and the available epidemiological studies are suggestive of a link between chronic health effects in humans and long-term ozone exposure. And most recently, a study by McDonnell et al. (1999) carefully measured ozone exposure over 15 years, and found ozone exposure was linked to the onset of asthma in adult males.

The McDonnell et al. (1999) study used the same cohort of Seventh-Day Adventists as Abbey et al. (1991; 1993), and examined the association between air pollution and the onset of asthma in adults between 1977 and 1992. Males who did not report doctor-diagnosed asthma in 1977, but reported it in 1987 or 1992, had significantly higher ozone exposures, controlling for other covariates; no significant effect was found between ozone exposure and asthma in females. No significant effect was reported for females or males due to exposure to PM, NO_2, SO_2, or SO_4.

Valuing Chronic Asthma

Two studies have estimated WTP to avoid chronic asthma in adults. Blumenschein and Johannesson (1998) used two different contingent valuation (CV) methods, the dichotomous choice method and a bidding game, to estimate mean willingness to pay for a cure for asthma. The mean WTP elicited from the bidding game was $189 per month, or $2,268 per year (in 1996$). The mean WTP elicited from the dichotomous choice approach was $343 per month, or $4,116 per year (in 1996$). Using $2,268 per year, a five percent discount rate, and 1997 life expectancies for males in the United States (National Center for Health Statistics, 1999, Table 5), the present discounted value of the stream of annual WTPs is about $35,000 (in 1997 $).

O'Conor and Blomquist (1997) estimated WTP to avoid chronic asthma from estimates of risk-risk tradeoffs. Combining the risk-risk tradeoffs with a statistical value of life, the annual value of avoiding asthma can be derived. Assuming a value of a statistical life of $6 million, they derived an annual WTP to avoid asthma of $1500 (O'Connor et al., 1997, p. 677). For a value of a statistical life of $5,894,400 (in 1997 $), the corresponding implied annual value of avoiding chronic asthma, based on O'Conor and Blomquist would be $1,474. Assuming a five percent discount rate and 1997 life expectancies for males in the United States, the present discounted value of the stream of annual WTPs would be about $22,000 (in 1997 $).

Following the method used for the §812 Prospective analysis, the uncertainty surrounding the WTP to avoid a case of chronic asthma among adult males was characterized by a triangular distribution on the range determined by the two WTP estimates. The range used in the §812 Prospective analysis was [$19,000, $30,000], centered at $25,000 (in 1990 $). In the current analysis these dollar values are converted to 1997 $ using the CPI-U for "all items."

4.3 HOSPITAL ADMISSIONS

We estimate impact of ozone and PM on both respiratory and cardiovascular hospital admissions. In addition, we estimate the impact of these pollutants on emergency visits for asthma.

4.3.1 Respiratory and Cardiovascular Hospital Admissions

Respiratory and cardiovascular hospital admissions are the two broad categories of hospital admissions that have been related to exposure to PM and ozone. For both PM-related and ozone-related hospital admissions there are multiple epidemiological studies that have estimated C-R functions that can be used in this analysis. The respiratory and the cardiovascular hospital admissions studies are listed in Exhibits 4-8 and 4-9, respectively. (Again, Appendices B and C provide details on each study.) Although the benefits associated with respiratory and cardiovascular hospital admissions are estimated separately in the analysis, the methods used to estimate changes in incidence and to value those changes are the same for both broad categories of hospital admissions. The two categories of hospital admissions are therefore discussed together in this section.

Although separate analyses are carried out for PM-related and ozone-related hospital admissions, the method of pooling multiple studies is the same in both cases. To estimate the incidence and monetary value of avoided hospital admissions, we pool the incidences and the monetary values corresponding to the incidence estimates from a variety of U.S. and Canadian studies, using a random effects weighting procedure. These studies differ from each other in two important ways: (1) Some studies considered people of all ages while others considered only people ages 65 and older; and (2) The ICD codes included in studies of respiratory hospital admissions and air pollution vary substantially.

Exhibit 4-8 Respiratory Hospital Admission Studies

Location	Study	Endpoints Estimated (ICD code)	Pollutants Used in Final Model	Age of Study Population
Toronto, Canada	Burnett et al. (1997)	all respiratory (464-466, 480-486, 490-494, 496)	$PM_{10-2.5}$, O_3	all ages
Toronto, Canada	Burnett et al. (1999)	asthma (493); respiratory infection (464, 466, 480-487, 494); COPD (490-492, 496)	O_3, $PM_{10-2.5}$ (asthma); O_3, $PM_{2.5}$ (respiratory infection); O_3, $PM_{10-2.5}$ (COPD).	all ages
Toronto, Canada	Thurston et al. (1994)	all respiratory (466, 480-482, 485, 490-493)	O_3, $PM_{2.5}$	all ages
Minneapolis-St. Paul, MN	Moolgavkar et al. (1997)	pneumonia (480-487); COPD (490-496)	O_3, PM_{10} (pneumonia); O_3, PM_{10} (COPD)	>64
Minneapolis-St. Paul, MN	Schwartz (1994c)	pneumonia (480-486); COPD (490-496)	O_3, PM_{10} (pneumonia); PM_{10} (COPD)	>64
Birmingham, AL	Schwartz (1994a)	pneumonia (480-487); COPD (490-496)	PM_{10}	>64
Detroit, MI	Schwartz (1994b)	pneumonia (480-486); non-asthma COPD (491-492, 494-496)	O_3, PM_{10}	>64
Spokane, WA	Schwartz (1996)	all respiratory (460-519)	PM_{10}	>64
New Haven, CT	Schwartz (1995)	all respiratory (460-519)	O_3, PM_{10}	>64
Tacoma, WA	Schwartz (1995)	all respiratory (460-519)	O_3, PM_{10}	>64
Seattle, WA	Sheppard et al. (1999)	asthma (493)	$PM_{2.5}$	<65

Exhibit 4-9 Cardiovascular Hospital Admission Studies

Location	Study	Endpoints Estimated (ICD code)	Pollutants Used in Final Model	Age of Study Population
Toronto, Canada	Burnett et al. (1997)	cardiac (410-414, 427-428)	O_3, $PM_{10-2.5}$	all ages
Toronto, Canada	Burnett et al. (1999)	dysrhythmias (427);	$PM_{2.5}$, O_3	all ages
Detroit, MI	Schwartz and Morris (1995)	ischemic heart disease (410-414); congestive heart failure (428)	PM_{10}	>64
Eight U.S. counties 1/88-12/90	Schwartz (1999)	cardiovascular disease (390-429)	PM_{10}	>64
Tucson, AZ 1/88-12/90	Schwartz (1997)	cardiovascular disease (390-429)	PM_{10}	>64

The broadest classification includes ICD codes 460-519 (e.g., Schwartz, 1996). Other studies, however, considered only subsets of the broader classification. For example, Burnett et al. (1997) consider

ICD-9 codes 466, 480-486, 490-494, and 496. It is unclear what the correct set of ICD codes is. If the broadest category (460-519) is too broad, including respiratory illnesses that are not linked to air pollution, we would expect the estimated pollutant coefficients to be biased downward; however, they would be used in combination with a larger baseline incidence in estimating changes in respiratory hospital admissions associated with changes in pollutant concentrations. If the broadest category is correct (i.e., if all the respiratory illnesses included are actually associated with air pollution), then studies using only subsets of ICD codes within that category would presumably understate the change in respiratory hospital admissions. It is likely, however, that all the studies have included the most important respiratory illnesses, and that the impact of differences in the definition of "all respiratory illnesses" may be less than that of other study design characteristics. We therefore treat each study equally, at least initially, in the pooling process, assuming that each study gives a reasonable estimate of the impact of air pollution on respiratory hospital admissions. The pooling process involves several steps.

1. For each study, develop a study-specific distribution of pollutant coefficients.[28] If separate non-overlapping sets of illnesses were considered in the study, develop a distribution for each set.

The value of the pollutant coefficient in a C-R function is estimated. Because of the statistical uncertainty surrounding the estimated coefficient, the C-R function is uncertain. We assume a normal distribution of the pollutant coefficient in the C-R function, with mean equal to the estimated coefficient reported in the study and standard deviation equal to the reported standard error of that estimate. If separate models were estimated for separate non-overlapping sets of illnesses (e.g., Burnett et al., 1999) estimated three separate models: one for asthma (ICD code 493), one for "respiratory infection" (ICD codes 464, 466, 480-487, and 494), and one for COPD (ICD codes 490-492, 496)), we develop a distribution of coefficients for each non-overlapping hospital admissions category.

2. For each study, develop a distribution of unit monetary values. If separate non-overlapping sets of respiratory illnesses were considered in the study, develop a distribution of unit monetary values for each set.

The monetary value of an avoided hospital admission depends on the particular type of illness (i.e., the ICD code) and the length of stay in the hospital, which itself varies with the type of admission. The monetary value of a set of hospital admissions (i.e., a set of ICD codes) is estimated as a weighted average of the individual ICD-code-specific values in the set. The valuation of hospital admissions is described more fully below.

3. For each study, develop a distribution of incidence changes and a distribution of monetary benefits resulting from a given change in pollutant concentrations.

On each iteration of a Monte Carlo procedure, for each non-overlapping hospital admissions category considered in the study,

- we randomly select a pollutant coefficient from the distribution of coefficients.
- Given the coefficient and the pollutant change, we calculate the incidence change.
- We randomly select a unit dollar value from the corresponding dollar distribution;
- The benefit is the product of the incidence change and the unit dollar value.

[28] "Pollutant" can refer either to PM or to ozone.

If the study has considered several non-overlapping hospital admissions categories, we sum the incidences and the dollar benefits across categories. For example, we estimated and summed the incidences for the three separate models estimated by Burnett et al. (1999). A series of many (e.g., 5000) iterations therefore produces (1) a series (distribution) of incidence changes for each non-overlapping hospital admissions category considered by the study as well as for all categories combined, and (2) a distribution of the dollar benefits associated with hospital admissions that would be predicted by the study.

4. Aggregate estimates across non-overlapping age categories.

Several studies estimated C-R functions for respiratory admissions for people ages 65 and older. One study, Sheppard et al. (1999), estimated a C-R function for asthma only for people under 65. Using a Monte Carlo procedure, we aggregate the results from the Sheppard study with those from each of the over-65 respiratory admissions studies.

5. Pool estimates of respiratory hospital admissions changes.

The study-specific incidence estimates are then pooled using a random effects pooling procedure, as described above. The study-specific dollar benefits estimates are similarly pooled.

Valuing Respiratory and Cardiovascular Hospital Admissions

Society's WTP to avoid a hospital admission includes medical expenses, lost work productivity, the non-market costs of treating illness (i.e., air, water and solid waste pollution from hospitals and the pharmaceutical industry), and the pain and suffering of the affected individual as well as of that of relatives, friends, and associated caregivers.[29]

Because medical expenditures are to a significant extent shared by society, via medical insurance, Medicare, etc., the medical expenditures actually incurred by the individual are likely to be less than the total medical cost to society. The total value to society of an individual's avoidance of hospital admission, then, might be thought of as having two components: (1) the cost of illness (COI) to society, including the total medical costs plus the value of the lost productivity, as well as (2) the WTP of the individual, as well as that of others, to avoid the pain and suffering resulting from the illness.

In the absence of estimates of social WTP to avoid hospital admissions for specific illnesses (components 1 plus 2 above), estimates of total COI (component 1) are typically used as conservative (lower bound) estimates. Because these estimates do not include the value of avoiding the pain and suffering resulting from the illness (component 2), they are biased downward. Some analyses adjust COI estimates upward by multiplying by an estimate of the ratio of WTP to COI, to better approximate total WTP. Other analyses have avoided making this adjustment because of the possibility of over-adjusting --

[29] Some people take action to avert the negative impacts of pollution. While the costs of successful averting behavior should be added to the sum of the health-endpoint-specific costs when estimating the total costs of pollution, these costs are not associated with any single health endpoint It is possible that in some cases the averting action was not successful, in which case it might be argued that the cost of the averting behavior should be added to the other costs listed (for example, it might be the case that an individual incurs the costs of averting behavior and in addition incurs the costs of the illness that the averting behavior was intended to avoid). Because averting behavior is generally not taken to avoid a particular health problem (such as a hospital admission for respiratory illness), but instead is taken to avoid the entire collection of adverse effects of pollution, it does not seem reasonable to ascribe the entire costs of averting behavior to any single health endpoint.

that is, possibly replacing a known downward bias with an upward bias. The previous RIAs for PM and ozone, as well as the revised RIA for ozone and PM NAAQS, did adjust the COI estimate upward. The COI values used in this benefits analysis will not be adjusted to better reflect the total WTP.

Following the method used in the §812 analysis (U.S. EPA, 1999a), ICD-code-specific COI estimates used in our analysis consist of two components: estimated hospital charges and the estimated opportunity cost of time spent in the hospital (based on the average length of a hospital stay for the illness). The opportunity cost of a day spent in the hospital is estimated as the value of the lost daily wage, regardless of whether or not the individual is in the workforce. This is estimated at $102 (U.S. Bureau of the Census, 1992).

For all hospital admissions included in this analysis, estimates of hospital charges and lengths of hospital stays were based on discharge statistics provided by Elixhauser et al. (1993). The total COI for an ICD-code-specific hospital stay lasting n days, then, would be estimated as the mean hospital charge plus $102*n. Most respiratory hospital admissions categories considered in epidemiological studies consisted of sets of ICD codes. The unit dollar value for the set of ICD codes was estimated to be a weighted average of the ICD-code-specific values, where the weights are the relative frequencies of hospital discharges (in Elixhauser et al. (1993)) of each ICD code in the set. The study-specific values for valuing respiratory and cardiovascular hospital admissions are shown in Exhibits 4-10 and 4-11, respectively.

Exhibit 4-10 Unit Values for Respiratory Hospital Admissions

Study	Endpoints Estimated (ICD code)	COI [a] (1997 $)
Burnett et al. (1997) Toronto, Canada	all respiratory (464-466, 480-486, 490-494, 496)	$ 9,914
Burnett et al. (1999) Toronto, Canada	asthma (493)	$ 6,301
	respiratory infection (464, 466, 480-487, 494)	$ 10,720
	COPD (490-492, 496)	$ 10,479
Thurston et al. (1994) Toronto, Canada	all respiratory (466, 480-482, 485, 490-493)	$ 9,652
Moolgavkar et al. (1997) Minneapolis-St. Paul, MN	pneumonia (480-487)	$ 11,429
	COPD (490-496)	$ 8,634
Schwartz (1994c) Minneapolis-St. Paul, MN	pneumonia (480-486)	$ 11,571
	COPD (490-496)	$ 8,634
Schwartz (1994a) Birmingham, AL	pneumonia (480-487)	$ 11,429
	COPD (490-496)	$ 8,634
Schwartz (1994b) Detroit, MI	pneumonia (480-486)	$ 11,571
	non-asthma COPD (491-492, 494-496)	$ 11,893
Schwartz (1996) Spokane, WA	all respiratory (460-519)	$ 10,326
Schwartz (1995) New Haven, CT	all respiratory (460-519)	$ 10,326
Schwartz (1995) Tacoma, WA	all respiratory (460-519)	$ 10,326
Sheppard et al. (1999) Seattle, WA	asthma (493)	$ 6,301

[a] The unit value for a group of ICD-9 codes is the weighted average of ICD-9 code-specific values, from Elixhauser et al. (1993). The weights are the relative frequencies of hospital discharges in Elixhauser et al. for each ICD-9 code in the group. The monetized benefits of non-overlapping endpoints within each study were aggregated. Monetized benefits for asthma among people age <65 (Sheppard et al., 1999) were aggregated with the benefits in studies of people age >64.

Exhibit 4-11 Unit Values for Cardiovascular Hospital Admissions

Study	Endpoints Estimated (ICD code)	COI [a] (1997 $)
Burnett et al. (1997) Toronto, Canada	cardiac (410-414, 427-428)	$ 13,430
Burnett et al. (1999) Toronto, Canada	dysrhythmias (427)	$ 6,483
Schwartz and Morris (1995) Detroit, MI	ischemic heart disease (410-414)	$ 16,142
	congestive heart failure (428)	$ 11,933
Schwartz (1999) Eight U.S. counties 1/88-12/90	cardiovascular disease (390-429)	$ 13,440
Schwartz (1997) Tucson, AZ 1/88-12/90	cardiovascular disease (390-429)	$ 13,440

[a] The unit value for a group of ICD-9 codes is the weighted average of ICD-9 code-specific values, from Elixhauser et al. (1993). The weights are the relative frequencies of hospital discharges in Elixhauser et al. for each ICD-9 code in the group.

The uncertainty surrounding cost-of-illness estimates for hospital admissions was based on the estimated means and standard errors of those means for hospital charges as reported in Elixhauser et al. (1993). For a hospital admission defined by a single ICD code (e.g., hospital admissions for congestive heart failure -- ICD-9 code 428), the uncertainty distribution of cost was characterized as a normal distribution with mean equal to the mean hospital charge for that ICD code and standard deviation equal to the standard error of that mean, as reported in Elixhauser et al. (1993).

For a hospital admission endpoint defined by a group of ICD codes, the uncertainty distribution of cost was defined by considering the cost to be a linear combination of the ICD code-specific costs, where the coefficient for each ICD code-specific cost is the relative frequency of hospital discharges for that ICD code in the group. The cost of a hospital admission for an illness category defined by a group of ICD codes (e.g., cardiovascular disease, defined as ICD codes 390-429), Y, is given by:

$$Y = \sum_{i=1}^{n} a_i X_i$$

where X_i is the hospital charge associated with the i^{th} ICD code in the group, and a_i is the relative frequency (in Elixhauser et al. (1993)) of hospital discharges for the i^{th} ICD code in the group. If each of the X's is distributed as a normal random variable, then Y is also a normal random variable, with mean equal to:

$$\sum_{i=1}^{n} a_i mean(X_i)$$

and variance equal to

$$\sum_{i=1}^{n} a_i^2 [s.e.(X_i)]^2 \quad .$$

The standard deviation of the distribution of Y would just be the square root of the variance.

4.3.2 Asthma-Related Emergency Room (ER) Visits

We use four C-R functions to estimate the effects of PM and ozone exposure to asthma-related ER visits. Three predict ozone-related asthma ER visits, while a fourth predicts asthma-related ER visits based on exposures to PM. Ozone-related asthma ER visits are based on epidemiological studies by Cody et al. (1992), Weisel et al. (1995), and Stieb et al. (1996). The first two studies, Cody et al. and Weisel et al., were conducted in Northern New Jersey. The Cody et al. study examined asthma-related ER visits over a 16 month period between May, 1988 and August, 1989, and found that ozone was linked to asthma-related ER visits. No significant effect was seen for PM_{10} or SO_2. Using a one-pollutant model, Weisel et al. also found ozone linked to asthma-related ER visits in an all-age 1990 population for eight New Jersey counties. Finally, Stieb et al. examined asthma-related ER visits over an eight year period from May through September in St. John, New Brunswick, Canada. Ozone was linked to ER visits within the all-ages population, especially when ozone levels exceeded 75 ppb. No significant effect was seen for the other pollutants.

Schwartz et al. (1993) failed to find a significant relationship between asthma-related ER visits and ozone. In this study of Seattle residents, Schwartz et al. instead found PM_{10} to be significantly related to asthma-related ER visits. The four studies are listed in Exhibit 4-12 below.

Exhibit 4-12 Asthma-Related Emergency Room Visit Studies

Location	Study	Pollutants Used in Final Model	Study Population
central and northern NJ	Cody et al. (1992)	O_3	all ages
central and northern NJ	Weisel et al. (1995)	O_3	all ages
Seattle, WA	Schwartz et al. (1993)	PM_{10}	<65
St. John, New Brunswick, Canada	Stieb et al. (1996)	O_3	all ages

Because we are estimating ER visits as well as hospital admissions for asthma, we must avoid counting twice the ER visits for asthma that are subsequently admitted to the hospital. To avoid double-counting, the baseline incidence rate for emergency room visits is adjusted by subtracting the percentage of patients that are admitted into the hospital. Three studies provide some information to do this: Richards et al. (1981, p. 350) reported that 13% of children's ER visits ended up as hospital admissions; Lipfert (1993, p. 230) reported that ER visits (for all causes) are two to five times more frequent than hospital admissions; Smith et al. (1997, p. 789) reported 445,000 asthma-related hospital admissions in 1987 and 1.2 million asthma ER visits. The study by Smith et al. seems the most relevant since it is a national study and looks at all age groups. Assuming that air-pollution related hospital admissions first pass through the ER, the reported incidence rates suggest that 37% (=445,000/1,200,000) of ER visits are subsequently admitted to the hospital, or that ER visits for asthma occur 2.7 times as frequently as hospital admissions for asthma. The baseline incidence of asthma ER visits is therefore taken to be 2.7 times the baseline incidence of

hospital admissions for asthma. To avoid double-counting, however, only 63% of the resulting change in asthma ER visits associated with a given change in pollutant concentrations is counted in the ER visit incidence change.

Valuing Asthma-Related Emergency Room (ER) Visits

The value of an avoided asthma-related ER visit was based on national data reported in Smith et al. (1997). Smith et al. reported that there were approximately 1.2 million asthma-related ER visits made in 1987, at a total cost of $186.5 million, in 1987$. The average cost per visit was therefore $155 in 1987$, or $279.55 in 1997 $ (using the CPI-U for medical care to adjust to 1997 $). The uncertainty surrounding this estimate, based on the uncertainty surrounding the number of ER visits and the total cost of all visits reported by Smith et al. was characterized by a triangular distribution centered at $279.55, on the interval [$207.50, $387.63].

4.4 ACUTE ILLNESSES AND SYMPTOMS NOT REQUIRING HOSPITALIZATION

We consider in this section a number of acute symptoms that do not require hospitalization, such as acute bronchitis, and upper and lower respiratory symptoms. Several of these illnesses and symptoms were considered in the §812 Prospective analysis as well. The unit values and the uncertainty distributions for those acute illnesses and symptoms that were also considered in the §812 Prospective analysis were obtained by adjusting the unit values used in that analysis from 1990 $ to 1997 $ by multiplying by 1.228 (based on the CPI-U for "all items").

4.4.1 Acute Bronchitis

Dockery et al. (1996) examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a study of 13,369 children ages 8-12 living in 24 communities in U.S. and Canada. Health data were collected in 1988-1991, and single-pollutant models were used in the analysis to test a number of measures of particulate air pollution. Dockery et al. found that annual level of sulfates and particle acidity were significantly related to bronchitis, and $PM_{2.5}$ and PM_{10} were marginally significantly related to bronchitis.

Valuing Acute Bronchitis

Estimating WTP to avoid a case of acute bronchitis is difficult for several reasons. First, WTP to avoid acute bronchitis itself has not been estimated. Estimation of WTP to avoid this health endpoint therefore must be based on estimates of WTP to avoid symptoms that occur with this illness. Second, a case of acute bronchitis may last more than one day, whereas it is a day of avoided symptoms that is typically valued. Finally, the C-R function used in the benefit analysis for acute bronchitis was estimated for children, whereas WTP estimates for those symptoms associated with acute bronchitis were obtained from adults.

With these caveats in mind, the values used for acute bronchitis in this analysis were obtained by adjusting the values used in the §812 Prospective analysis from 1990 $ to 1997 $ by multiplying by 1.228. WTP to avoid a case of acute bronchitis was estimated as the midpoint between a low estimate and a high

estimate. The low estimate is the sum of the midrange values recommended by IEc (1994) for two symptoms believed to be associated with acute bronchitis: coughing and chest tightness. The high estimate was taken to be twice the value of a minor respiratory restricted activity day. The unit value is the midpoint between the low and high estimates. The low, high, and midpoint estimates used in the §812 Prospective analysis were $13, $77, and $45, respectively, in 1990 $. The corresponding values in 1997 $ are $15.96, $94.56, and $55.26, respectively.

4.4.2 Upper Respiratory Symptoms (URS)

Using logistic regression, Pope et al. (1991) estimated the impact of PM_{10} on the incidence of a variety of minor symptoms in 55 subjects (34 "school-based" and 21 "patient-based") living in the Utah Valley from December 1989 through March 1990. The children in the Pope et al. study were asked to record respiratory symptoms in a daily diary, and the daily occurrences of URS and LRS, as defined above, were related to daily PM_{10} concentrations. Pope et al. describe URS as consisting of one or more of the following symptoms: runny or stuffy nose; wet cough; and burning, aching, or red eyes. Levels of ozone, NO_2, and SO_2 were reported low during this period, and were not included in the analysis.

The sample in this study is relatively small and is most representative of the asthmatic population, rather than the general population. The school-based subjects (ranging in age from 9 to 11) were chosen based on "a positive response to one or more of three questions: ever wheezed without a cold, wheezed for 3 days or more out of the week for a month or longer, and/or had a doctor say the 'child has asthma' (Pope et al., 1991, p. 669)." The patient-based subjects (ranging in age from 8 to 72) were receiving treatment for asthma and were referred by local physicians. Regression results for the school-based sample (Pope et al., 1991, Table 5) show PM_{10} significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM_{10} effect. The results from the school-based sample are used here.

Valuing URS

Willingness to pay to avoid a day of URS is based on symptom-specific WTPs to avoid those symptoms identified by Pope et al. as part of the URS complex of symptoms. Three contingent valuation (CV) studies have estimated WTP to avoid various morbidity symptoms that are either within the URS symptom complex defined by Pope et al. (1991) or are similar to those symptoms identified by Pope et al. In each CV study, participants were asked their WTP to avoid a day of each of several symptoms. The WTP estimates corresponding to the morbidity symptoms valued in each study are presented in Exhibit 4-13. The three individual symptoms listed in Exhibit 4-13 that were identified as most closely matching those listed by Pope, et al. for URS are cough, head/sinus congestion, and eye irritation, corresponding to "wet cough," "runny or stuffy nose," and "burning, aching or red eyes," respectively. A day of URS could consist of any one of the seven possible "symptom complexes" consisting of at least one of these three symptoms. Using the symptom symbols in Exhibit 4-13, these seven possible symptom complexes are presented in Exhibit 4-14. It is assumed that each of these seven URS complexes is equally likely.[30] The point estimate of MWTP to avoid an occurrence of URS is just an average of the seven estimates of MWTP for the different URS complexes – $18.70, or about $19 in 1990 $. This is $23.33 (=$19*1.228)

[30] With empirical evidence, we could presumably improve the accuracy of the probabilities of occurrence of each type of URS. Lacking empirical evidence, however, a uniform distribution seems the most reasonable "default" assumption.

in 1997 $. In the absence of information surrounding the frequency with which each of the seven types of URS occurs within the URS symptom complex, an uncertainty analysis for WTP to avoid a day of URS is based on a continuous uniform distribution of MWTPs in Exhibit 4-14, with a range of [$7, $33], or [$8.60, $40.52] in 1997 $.

Exhibit 4-13 Median WTP Estimates and Derived Midrange Estimates (in 1997 $)

Symptom [a]	Dickie et al. (1987)	Tolley et al. (1986)	Loehman et al. (1979)	Mid-Range Estimate
Throat congestion	4.63	20.08	-	12.28
Head/sinus congestion	5.40	21.63	10.07	12.28
Coughing	1.55	17.00	6.12	8.60
Eye irritation	-	19.30	-	19.30
Headache	1.55	30.90	-	12.28
Shortness of breath	0.00	-	12.98	6.14
Pain upon deep inhalation (PDI)	5.42	-	-	5.42
Wheeze	3.09	-	-	3.09
Coughing up phlegm	3.38 [b]	-	-	3.38
Chest tightness	7.74	-	-	7.74

[a] All estimates are WTP to avoid one day of symptom. Midrange estimates were derived by IEc (1993).

[b] 10% trimmed mean.

Exhibit 4-14 Estimates of MWTP to Avoid Upper Respiratory Symptoms (1997 $)

Symptom Combinations Identified as URS by Pope et al. (1991)	MWTP to Avoid Symptom(s)
Coughing	$8.60
Head/Sinus Congestion	$12.28
Eye Irritation	$19.30
Coughing, Head/Sinus Congestion	$20.88
Coughing, Eye Irritation	$27.90
Head/Sinus Congestion, Eye Irritation	$31.58
Coughing, Head/Sinus Congestion, Eye Irritation	$40.18
	Average: $22.96

Based on values reported in Exhibit 4-13.

It is worth emphasizing that what is being valued here is URS *as defined by Pope et al. (1991).* While other definitions of URS are certainly possible, this definition of URS is used in this benefit analysis because it is the incidence of this specific definition of URS that has been related to PM exposure by Pope et al.

4.4.3 Lower Respiratory Symptoms (LRS)

Schwartz et al. (1994) used logistic regression to link lower respiratory symptoms in children with SO_2, NO_2, ozone, PM_{10}, $PM_{2.5}$, sulfate and H^+ (hydrogen ion). Children were selected for the study if they were exposed to indoor sources of air pollution: gas stoves and parental smoking. The study enrolled 1,844 children into a year-long study that was conducted in different years (1984 to 1988) in six cities. The students were in grades two through five at the time of enrollment in 1984. By the completion of the final study, the cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

In single pollutant models SO_2, NO_2, $PM_{2.5}$, and PM_{10} were significantly linked to cough. In two-pollutant models, PM_{10} had the most consistent relationship with cough; ozone was marginally significant, controlling for PM_{10}. In models for upper respiratory symptoms, they reported a marginally significant association for PM_{10}. In models for lower respiratory symptoms, they reported significant single-pollutant models, using SO_2, O_3, $PM_{2.5}$, PM_{10}, SO_4, and H^+.

Valuing LRS

The method for deriving a point estimate of mean WTP to avoid a day of LRS is the same as for URS. Schwartz et al. (1994, p. 1235) define LRS as at least two of the following symptoms: cough, chest pain, phlegm, and wheeze. The symptoms for which WTP estimates are available that reasonably match those listed by Schwartz et al. for LRS are cough (C), chest tightness (CT), coughing up phlegm (CP), and wheeze (W). A day of LRS, as defined by Schwartz et al., could consist of any one of the 11 combinations of at least two of these four symptoms, as displayed in Exhibit 4-15.[31]

[31] Because cough is a symptom in some of the URS clusters as well as some of the LRS clusters, there is the possibility of a very small amount of double counting – if the same individual were to have an occurrence of URS which included cough and an occurrence of LRS which included cough *both on exactly the same day.* Because this is probably a very small probability occurrence, the degree of double counting is likely to be very minor. Moreover, because URS is applied only to asthmatics ages 9-11 (a very small population), the amount of potential double counting should be truly negligible.

Exhibit 4-15 Estimates of MWTP to Avoid Lower Respiratory Symptoms (1997 $)

Symptom Combinations Identified as LRS by Schwartz et al. (1994)	MWTP to Avoid Symptom(s)
Coughing, Chest Tightness	$16.33
Coughing, Coughing Up Phlegm	$11.97
Coughing, Wheeze	$11.69
Chest Tightness, Coughing Up Phlegm	$11.11
Chest Tightness, Wheeze	$10.83
Coughing Up Phlegm, Wheeze	$6.47
Coughing, Chest Tightness, Coughing Up Phlegm	$19.71
Coughing, Chest Tightness, Wheeze	$19.43
Coughing, Coughing Up Phlegm, Wheeze	$15.07
Chest Tightness, Coughing Up Phlegm, Wheeze	$14.21
Coughing, Chest Tightness, Coughing Up Phlegm, Wheeze	$22.80
	Average: $14.51

Based on values reported in Exhibit 4-13.

We assumed that each of the eleven types of LRS is equally likely.[32] The mean WTP to avoid a day of LRS as defined by Schwartz et al. (1994) is therefore the average of the mean WTPs to avoid each type of LRS, – $11.82, which rounds to $12.00. This is $14.74 (=1.228*$12.00) in 1997 $. This is the point estimate used in the benefit analysis for the dollar value for LRS as defined by Schwartz et al. The WTP estimates are based on studies which considered the value of a *day* of avoided symptoms, whereas the Schwartz et al. study used as its measure a *case* of LRS. Because a case of LRS usually lasts at least one day, and often more, WTP to avoid a day of LRS should be a conservative estimate of WTP to avoid a case of LRS.

In the absence of information about the frequency of each of the seven types of LRS among all occurrences of LRS, the uncertainty analysis for WTP to avoid a day of URS is based on a continuous uniform distribution of MWTPs in Exhibit 4-13, with a range of [$5, $19], or [$6.14, $23.33] in 1997 $. This is the same procedure as that used in the URS uncertainty analysis.

As with URS, it is worth emphasizing that what is being valued here is LRS *as defined by Schwartz et al. (1994)*. While other definitions of LRS are certainly possible, this definition of LRS is used in this benefit analysis because it is the incidence of this specific definition of LRS that has been related to PM exposure by Schwartz et al.

[32] As with URS, if we had empirical evidence we could improve the accuracy of the probabilities of occurrence of each type of LRS. Lacking empirical evidence, however, a uniform distribution seems the most reasonable "default" assumption.

Issues in the Valuation of URS and LRS

The point estimates derived for mean WTP to avoid a day of URS and a case of LRS are based on the assumption that WTPs are additive. For example, if WTP to avoid a day of cough is $8.60, and WTP to avoid a day of shortness of breath is $6.14, then WTP to avoid a day of both cough and shortness of breath is $14.74. If there are no synergistic effects among symptoms, then it is likely that the marginal utility of avoiding symptoms decreases with the number of symptoms being avoided. If this is the case, adding WTPs would tend to overestimate WTP for avoidance of multiple symptoms. However, there may be synergistic effects– that is, the discomfort from two or more simultaneous symptoms may exceed the sum of the discomforts associated with each of the individual symptoms. If this is the case, adding WTPs would tend to underestimate WTP for avoidance of multiple symptoms. It is also possible that people may experience additional symptoms for which WTPs are not available, again leading to an underestimate of the correct WTP. However, for small numbers of symptoms, the assumption of additivity of WTPs is unlikely to result in substantive bias.

There are also three sources of uncertainty in the valuation of both URS and LRS: (1) an occurrence of URS or of LRS may be comprised of one or more of a variety of symptoms (i.e., URS and LRS are each potentially a "complex of symptoms"), so that what is being valued may vary from one occurrence to another; (2) for a given symptom, there is uncertainty about the mean WTP to avoid the symptom; and (3) the WTP to avoid an occurrence of multiple symptoms may be greater or less than the sum of the WTPs to avoid the individual symptoms.

Information about the degree of uncertainty from either the second or the third source is not available. The first source of uncertainty, however, is addressed because an occurrence of URS or LRS may vary in symptoms. For example, seven different symptom complexes that qualify as URS, as defined by Pope et al. (1991), were identified above. The estimates of MWTP to avoid these seven different kinds of URS range from $8.60 (to avoid an occurrence of URS that consists of only coughing) to $40.52 (to avoid an occurrence of URS that consists of coughing plus head/sinus congestion plus eye irritation). There is no information, however, about the frequency of each of the seven types of URS among all occurrences of URS.

Because of insufficient information to adequately estimate the distributions of the estimators of MWTP for URS and LRS, as a rough approximation, a continuous uniform distribution over the interval from the smallest point estimate to the largest is used. As was mentioned in the two previous sections, the interval for URS is [$8.60, $40.52], and for LRS, the interval is [$6.14, $23.33].

Alternatively, a discrete distribution of the seven unit dollar values associated with each of the seven types of URS identified could be used. This would provide a distribution whose mean is the same as the point estimate of MWTP. A continuous uniform distribution, however, is probably more reasonable than a discrete uniform distribution. The differences between the means of the discrete uniform distributions (the point estimates) and the means of the continuous uniform distributions are relatively small, as shown in Exhibit 4-16.

Exhibit 4-16 Comparison of the Means of Discrete and Continuous Uniform Distributions of MWTP Associated with URS and LRS (1990 $)

Health Endpoint	Mean of Discrete Uniform Distribution (Point Est.)	Mean of Continuous Uniform Distribution
URS (Pope et al., 1991)	18.70	19.86
LRS (Schwartz et al., 1994)	11.82	11.92

4.4.4 "Any of 19 Respiratory Symptoms" and Minor Restricted Activity Days (MRADs)

Two studies, one by Ostro and Rothschild (1989b) and the other by Krupnick et al. (1990), cover a variety of minor symptoms. To avoid double counting, we treat these two studies as alternative measures of the same health effect, and pool the incidence estimates. The method of pooling incidence and benefits estimates is the same as that used for hospital admissions and is described above in the section on respiratory and cardiovascular hospital admissions.

Ostro and Rothschild (1989b) estimated the impact of $PM_{2.5}$ on the incidence of minor restricted activity days (MRAD) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. We developed separate coefficients for each year in the analysis (1976-1981), which were then combined for use in this analysis. The coefficient used in the C-R function is a weighted average of the coefficients in Ostro (Ostro, 1987, Table IV) using the inverse of the variance as the weight.

Krupnick et al. (1990) estimated the impact of coefficient of haze (COH, a measure of particulate matter concentrations), ozone and SO_2 on the incidence of any of 19 symptoms or conditions in the adult population, ages 18 to 65.[33] They used a logistic regression model that takes into account whether a respondent was well or not the previous day. A key difference between this and the usual logistic model is that the model they used includes a lagged value of the dependent variable. This makes the derivation of a C-R function somewhat more complicated than the usual logistic regression.[34]

The presence of "any of 19 acute respiratory symptoms" is a somewhat subjective health effect used by Krupnick et al. (1990). Moreover, not all 19 symptoms are listed in the Krupnick et al. study. It is therefore not clear exactly what symptoms were included in the study. Even if all 19 symptoms were known, it is unlikely that WTP estimates could be obtained for all of the symptoms. Finally, even if all 19 symptoms were known and WTP estimates could be obtained for all 19 symptoms, the assumption of additivity of WTPs becomes tenuous with such a large number of symptoms. The likelihood that all 19 symptoms would occur simultaneously, moreover, is very small.

In addition to the overlapping health effects present in both of these endpoints, the Ostro and Rothschild (1989b) study, as well as the Krupnick et al. (1990) study, overlap with a smaller subset of

[33]Krupnick et al. (1990) list 13 specific "symptoms or conditions": head cold, chest cold, sinus trouble, croup, cough with phlegm, sore throat, asthma, hay fever, doctor-diagnosed ear infection, flu, pneumonia, bronchitis, and bronchiolitis. The other six symptoms or conditions are not specified.

[34]Details of the derivation of the C-R function based on the model used by Krupnick et al. (1990) are presented in Abt Associates (1999, p. A-40).

health effects predicted by other studies. In particular, two studies predict asthma attacks (Whittemore and Korn (1980)) and moderate or worse asthma (Ostro et al. (1991)) (both discussed later in the text). These endpoints are included in the array of health effects covered by the pooled "any of 19"/MRAD incidence estimate, and would thus constitute a double counting of benefits if included in the primary analysis. Instead, asthma attack incidence and moderate or worse asthma incidence are presented as supplemental calculations to the pooled incidence estimate of "any of 19 symptoms" and MRADs.

Valuing "Any of 19 Respiratory Symptoms"

The unit value and uncertainty distribution for "any of 19 respiratory symptoms" for this analysis were obtained by adjusting the (rounded) values in 1990 $ used in the §812 Prospective analysis to 1997 $ by multiplying by 1.228. Acute respiratory symptoms must be either upper respiratory symptoms or lower respiratory symptoms. In the absence of further knowledge about which of the two types of symptoms is more likely to occur among the "any of 19 acute respiratory symptoms," we assumed that they occur with equal probability. Because this health endpoint may also consist of combinations of symptoms, it was also assumed that there is some (smaller) probability that upper and lower respiratory symptoms occur together. To value avoidance of a day of "the presence of any of 19 acute respiratory symptoms" we therefore assumed that this health endpoint consists either of URS, or LRS, or both. We also assumed that it is as likely to be URS as LRS and that it is half as likely to be both together. That is, it was assumed that "the presence of any of 19 acute respiratory symptoms" is a day of URS with 40 percent probability, a day of LRS with 40 percent probability, and a day of both URS and LRS with 20 percent probability. Using the point estimates of WTP to avoid a day of URS and LRS derived above, the point estimate of WTP to avoid a day of "the presence of any of 19 acute respiratory symptoms" is:

$$(0.40)(\$18.70) + (0.40)(\$11.82) + (0.20)(\$18.70 + \$11.82) = \$18.31, \text{ or about } \$18 \text{ (1990 \$)} .$$

This is $22.10 (=$18*1.228) in 1997 $. Because this health endpoint is only vaguely defined, and because of the lack of information on the relative frequencies of the different combinations of acute respiratory symptoms that might qualify as "any of 19 acute respiratory symptoms," the unit dollar value derived for this health endpoint must be considered only a rough approximation.

The sources of uncertainty in the valuation of LRS and URS described above similarly exist in the valuation of this health endpoint. In particular, (1) "the presence of any of 19 acute respiratory symptoms" may be comprised of one or more of a variety of symptoms, so that what is being valued may vary from one occurrence to another; (2) for a given symptom, there is uncertainty about the mean WTP to avoid the symptom; and (3) the WTP to avoid an occurrence of multiple symptoms may be greater or less than the sum of the WTPs to avoid the individual symptoms.

To characterize the uncertainty surrounding the estimated value of avoiding "any of 19 acute respiratory symptoms," we used the distributions described above for the input components, URS and LRS. On each iteration of a Monte Carlo procedure, URS was chosen with 40 percent probability, LRS was chosen with 40 percent probability and URS+LRS was chosen with 20 percent probability. Given the choice, a dollar value was randomly selected from the appropriate distribution. For example, if URS was selected, a dollar value was selected from the continuous uniform distribution for URS.

Valuing Minor Restricted Activity Days (MRADs)

The unit value and uncertainty distribution for MRADs for this analysis were obtained by adjusting the (rounded) values in 1990 $ used in the §812 Prospective analysis to 1997 $ by multiplying by 1.228. No studies are reported to have estimated WTP to avoid a minor restricted activity day (MRAD). However, IEc (1993) has derived an estimate of WTP to avoid a minor respiratory restricted activity day (MRRAD), using WTP estimates from Tolley et al. (1986) for avoiding a three-symptom combination of coughing, throat congestion, and sinusitis. This estimate of WTP to avoid a MRRAD, so defined, is $38.37 (1990 $), or about $38. Although Ostro and Rothschild (1989b) estimated the relationship between $PM_{2.5}$ and MRADs, rather than MRRADs (a component of MRADs), it is likely that most of the MRADs associated with exposure to $PM_{2.5}$ are in fact MRRADs. For the purpose of valuing this health endpoint, then, we assumed that MRADs associated with PM exposure may be more specifically defined as MRRADs, and therefore used the estimate of mean WTP to avoid a MRRAD.

Any estimate of mean WTP to avoid a MRRAD (or any other type of restricted activity day other than WLD) will be somewhat arbitrary because the endpoint itself is not precisely defined. Many different combinations of symptoms could presumably result in some minor or less minor restriction in activity. Krupnick and Kopp (1988) argued that mild symptoms will not be sufficient to result in a MRRAD, so that WTP to avoid a MRRAD should exceed WTP to avoid any single mild symptom. A single severe symptom or a combination of symptoms could, however, be sufficient to restrict activity. Therefore WTP to avoid a MRRAD should, these authors argue, not necessarily exceed WTP to avoid a single severe symptom or a combination of symptoms. The "severity" of a symptom, however, is similarly not precisely defined; moreover, one level of severity of a symptom could induce restriction of activity for one individual while not doing so for another. The same is true for any particular combination of symptoms.

Given that there is inherently a substantial degree of arbitrariness in any point estimate of WTP to avoid a MRRAD (or other kinds of restricted activity days), the reasonable bounds on such an estimate must be considered. By definition, a MRRAD does not result in loss of work. WTP to avoid a MRRAD should therefore be less than WTP to avoid a WLD. At the other extreme, WTP to avoid a MRRAD should exceed WTP to avoid a single mild symptom. The highest IEc midrange estimate of WTP to avoid a single symptom is $15.72 (1990 $), or about $16, for eye irritation. The point estimate of WTP to avoid a WLD in the benefit analysis is $83 (1990 $). If all the single symptoms evaluated by the studies are not severe, then the estimate of WTP to avoid a MRRAD should be somewhere between $16 and $83. Because the IEc estimate of $38 falls within this range (and acknowledging the degree of arbitrariness associated with any estimate within this range), the IEc estimate is used as the mean of a triangular distribution centered at $38, ranging from $16 to $61. Adjusting to 1997 $, this is a triangular distribution centered at $46.66, ranging from $19.65 to $74.91.

4.4.5 Shortness of Breath

Using logistic regression, Ostro et al. (1995) estimated the impact of PM_{10}, ozone, NO_2, and SO_2 on the incidence of coughing, shortness of breath, and wheezing in 83 African-American asthmatic children aged 7-12 living in Los Angeles from August through September 1992. Regression results show both PM_{10} and ozone significantly linked to shortness of breath; the beginning of an asthma episode was also significantly linked to ozone. Results for single-pollutant models only were presented in the published paper.

Valuing Shortness of Breath

A point estimate of mean WTP to avoid a day of shortness of breath was derived as the mean of the median estimates from two studies that evaluated this symptom. The median estimate from Dickie et al. (1987), was $0.00; the median estimate from Loehman et al. (1979) was $10.57, or about $10.60 (1990 $). The mean of these two medians is $5.30, or $6.51 in 1997 $. In the absence of sufficient information to characterize the distribution of MWTP to avoid a day of shortness of breath, this distribution is roughly approximated by a continuous distribution on the interval from the low estimate to the high estimate – [$0.00, $10.60] in 1990 $, or [$0.00, $13.02] in 1997 $.

4.4.6 Work Loss Days (WLD)

Ostro (1987) estimated the impact of $PM_{2.5}$ on the incidence of work-loss days (WLDs), restricted activity days (RADs), and respiratory-related RADs (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average $PM_{2.5}$ levels were significantly linked to work-loss days, RADs, and RRADs, however there was some year-to-year variability in the results. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function used here is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight.

Valuing WLD

Willingness to pay to avoid the loss of one day of work was estimated by dividing the median weekly wage for 1990 (U.S. Bureau of the Census, 1992) by five (to get the median daily wage). This values the loss of a day of work at the national median wage for the day lost. To account for regional variations in median wages, the national daily median wage was adjusted on a county-by-county basis using a factor based on the ratio of national median household income divided by each county's median income. Each county's income-adjusted willingness to pay to avoid the loss of one day of work was then used to value the number of work loss days attributed to that county. Valuing the loss of a day's work at the wages lost is consistent with economic theory, which assumes that an individual is paid exactly the value of his labor.[35]

The use of the median rather than the mean, however, requires some comment. If all individuals in society were equally likely to be affected by air pollution to the extent that they lose a day of work because of it, then the appropriate measure of the value of a work loss day would be the mean daily wage. It is highly likely, however, that the loss of work days due to pollution exposure does not occur with equal probability among all individuals, but instead is more likely to occur among lower income individuals than among high income individuals. It is probable, for example, that individuals who are vulnerable enough to the negative effects of air pollution to lose a day of work as a result of exposure tend to be those with generally poorer health care. Individuals with poorer health care have, on average, lower incomes. To estimate the average lost wages of individuals who lose a day of work because of exposure to PM

[35] The estimate of the value of work loss days avoided could be improved if, instead of a single national wage rate, state-specific or county-specific wage rates were used.

pollution, then, would require a weighted average of all daily wages, with higher weights on the low end of the wage scale and lower weights on the high end of the wage scale. Because the appropriate weights are not known, however, the median wage was used rather than the mean wage. The median is more likely to approximate the correct value than the mean because means are highly susceptible to the influence of large values in the tail of a distribution (in this case, the small percentage of very large incomes in the United States), whereas the median is not susceptible to these large values. The median daily wage in 1990 was $83, or $101.92 in 1997 $. This is the value that was used to represent work loss days (WLD). An uncertainty distribution for this endpoint was unavailable, therefore the same central estimate ($101.92) was used to value incidence changes at the fifth, mean, and ninety-fifth percentiles.

4.4.7 Worker Productivity

To monetize benefits associated with increased worker productivity resulting from improved ozone air quality, we used information reported in Crocker and Horst (1981) and summarized in EPA (1994). Crocker and Horst examined the impacts of ozone exposure on the productivity of outdoor citrus workers. The study measured productivity impacts as the change in income associated with a change in ozone exposure, given as the elasticity of income with respect to ozone concentration (-0.1427).[36] The reported elasticity translates a ten percent reduction in ozone to a 1.4 percent increase in income. Given the median daily income for outdoor workers engaged in strenuous activity reported by the 1990 U.S. Census, $89.64 per day (1997 $), a ten percent reduction in ozone yields about $1.26 in increased daily wages. The median daily income for outdoor workers is a national estimate, however. We adjust this estimate to reflect regional variations in income using a factor based on the ratio of national median household income divided by a county's median household income. No information was available for quantifying the uncertainty associated with the central valuation estimate. Therefore, no uncertainty analysis was conducted for this endpoint.

4.4.8 Supplemental Endpoints: Acute Illnesses And Symptoms Not Requiring Hospitalization

The benefits associated with several endpoints are estimated separately but are not included in the total benefits estimates because of the possibility of double counting of benefits. Two studies estimate the incidence of asthma (which overlap with the pooled measure of "any of 19 symptoms" and MRADs) and one study estimates the incidence of restricted activity days (which overlaps with measures of work loss days and MRADs).

Asthma Attacks

Whittemore and Korn (1980) examined the relationship between air pollution and asthma attacks in a survey of 443 children and adults, living in six communities in southern California during three 34-week periods in 1972-1975. The analysis focused on TSP and ozone. Respirable PM, NO_2, SO_2 were highly correlated with TSP and excluded from the analysis. In a two pollutant model, daily levels of both TSP and O_x were significantly related to reported asthma attacks. The value of an asthma attack is assumed to be the same as for a day in which asthma is moderate or worse.

[36] The relationship estimated by Crocker and Horst (1981) between wages and ozone is a log-log relationship. Therefore the elasticity of wages with respect to ozone is a constant, equal to the coefficient of log ozone in the model.

Valuing Asthma Attacks

The value of avoiding an asthma attack is estimated as the mean of four WTP estimates obtained in a study by Rowe and Chestnut (1986). The four WTP estimates correspond to four severity definitions of a "bad asthma day." The mean of the four average WTPs is $32 (1990 $), or $39.30 in 1997 $. The uncertainty surrounding this estimate was characterized by a continuous uniform distribution on the range defined by the lowest and highest of the four average WTP estimates from Rowe and Chestnut, [$12, $54], or [$14.74, $66.31] in 1997 $.

Moderate or Worse Asthma

Ostro et al. (1991) examined the effect of air pollution on asthmatics, ages 18 to 70, living in Denver, Colorado from December 1987 to February 1988. The respondents in this study were asked to record daily a subjective rating of their overall asthma status each day (0=none, 1=mild, 2=moderate, 3=severe, 4=incapacitating). Ostro et al. then examined the relationship between moderate (or worse) asthma and H^+, sulfate, SO_2, $PM_{2.5}$, estimated $PM_{2.5}$, PM_{10}, nitrate, and nitric acid. Daily levels of H^+ were linked to cough, asthma, and shortness of breath. $PM_{2.5}$ was linked to asthma. SO_2 was linked to shortness of breath. No effects were seen for other pollutants.

Valuing Moderate or Worse Asthma

The unit value and uncertainty distribution for moderate or worse asthma were assumed to be the same as for an asthma attack (see above), based on four WTP estimates from Rowe and Chestnut (1986). The mean of the four average WTPs is $32 (1990 $), or $39.30 in 1997 $. The uncertainty surrounding this estimate was characterized by a continuous uniform distribution on the range defined by the lowest and highest of the four average WTP estimates from Rowe and Chestnut, [$12, $54], or [$14.74, $66.31] in 1997 $.

Although subjects' assessment of what constitutes a "bad asthma day" varied considerably in the Rowe and Chestnut (1986) study, the subjective assessment of an asthma day being bad is very similar to the subjective assessment of an asthma day being "of moderate or worse status" in the Ostro et al. (1991) study, in which subjects were also asked their subjective assessments.

Restricted Activity Days (RADs)

Ostro (1987) estimated the impact of $PM_{2.5}$ on the incidence of work-loss days (WLDs), restricted activity days (RADs), and respiratory-related RADs (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average $PM_{2.5}$ levels were significantly linked to work-loss days, RADs, and RRADs, however there was some year-to-year variability in the results. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function used here is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight.

The health effects included in the definition of RADs overlap with health effects included in both measures of work loss days and minor restricted activity days. To include both of these endpoints along with restricted activity days would lead to a double-counting of benefits, therefore restricted activity days are presented as a supplemental calculation of incidence only.

5 WELFARE BENEFITS

This analysis considers four types of benefits that are loosely termed "welfare" benefits. These include visibility improvements, reductions in agricultural crop damage, reduced household soiling, and reduced nitrogen deposition into estuaries. We consider each in turn.

5.1 VISIBILITY BENEFITS

Visibility degradation estimates used in this analysis are generated by the CRDM SR Matrix. Because these air quality-related changes in visibility are directly used in the benefits analysis, the methodology for predicting visibility changes is not discussed here. The visibility estimation methodology is described in detail in Pechan-Avanti (1999).

Economic benefits may result from two broad categories of visibility changes: (1) changes in "residential" visibility – i.e., the visibility in and around the locations where people live; and (2) changes in "recreational" visibility at Class I areas – i.e., visibility at Class I national parks and wilderness areas.[37] In this analysis, only recreational benefits are included in the primary presentation of benefits; residential benefits are presented as an alternative calculation of visibility benefits.

Within the category of recreational visibility, further distinctions have been made. There is evidence (Chestnut and Rowe, 1990) that an individual's WTP for improvements in visibility at a Class I area is influenced by whether it is in the region in which the individual lives, or whether it is somewhere else. In general people appear to be willing to pay more for visibility improvements at parks and wilderness areas that are "in-region" than at those that are "out-of-region." This is plausible, because people are more likely to visit, be familiar with, and care about parks and wilderness areas in their own part of the country.

To value estimated visibility changes, we are using an approach consistent with economic theory. Below we discuss an application of the Constant Elasticity of Substitution (CES) utility function approach[38] to value both residential visibility improvements and visibility improvements at Class I areas in the United States. This approach is based on the preference calibration method developed by Smith et al. (1999). The presentation of this methodology is organized as follows. The basic utility model is presented in Section 5.1.1. In Section 5.1.2 we discuss the measurement of visibility, and the mapping from environmental "bads" to environmental "goods." In Sections 5.1.3 and 5.1.4 we summarize the information that is available to estimate the parameters of the model corresponding to visibility at in-region and out-of-region Class I areas, and visibility in residential areas, respectively, and we describe the methods used to estimate these parameters. Section 5.1.5 synthesizes the results.

---

[37] Hereafter referred to as Class I areas, which are defined as areas of the country such as national parks, national wilderness areas, and national monuments that have been set aside under Section 162(a) of the Clean Air Act to receive the most stringent degree of air quality protection. Class I federal lands fall under the jurisdiction of three federal agencies, the National Park Service, the Fish and Wildlife Service, and the Forest Service.

[38] The Constant Elasticity of Substitution utility function has been chosen for use in this analysis due to its flexibility when illustrating the degree of substitutability present in various economic relationships (in this case, the tradeoff between income and improvements in visibility).

5.1.1 Basic Utility Model

We begin with a CES utility function in which a household derives utility from

(1) "all consumption goods," X,
(2) visibility in the residential area in which the household is located ("residential visibility"),[39]
(3) visibility at Class I areas in the same region as the household ("in-region recreational visibility"), and
(4) visibility at Class I areas outside the household's region ("out-of-region recreational visibility").

There are a total of six regions being considered, so there are 5 regions for which any household is out-of-region. The utility function of a household in the n^{th} residential area and the ith region of the country is:

$$U_{ni} = \left(X^r + qZ_n^r + \sum_{k=1}^{N_i} g_{ik} Q_{ik}^r + \sum_{j \neq i} \sum_{k=1}^{N_j} d_{jk} Q_{jk}^r \right)^{1/r} ,$$

$$q > 0, g_{ik} > 0, \forall i,k, d_{jk} > 0, \forall j,k, \; r \leq 1.$$

where

Z_n = the level of visibility in the nth residential area;
Q_{ik} = the level of visibility at the kth in-region park (i.e., the kth park in the ith region);
Q_{jk} = the level of visibility at the kth park in the jth region (for which the household is out-of-region), $j \neq i$;
N_i = the number of Class I areas in the ith region;
N_j = the number of Class I areas in the jth region (for which the household is out-of-region), $j \neq i$; and

θ, the γ's and δ's are parameters of the utility function corresponding to the visibility levels at residential areas, and at in-region and out-of-region Class I areas, respectively. In particular, the γ_{ik}'s are the parameters corresponding to visibility at in-region Class I areas; the δ_1's are the parameters corresponding to visibility at Class I areas in region 1 (California), if $i \neq 1$; the δ_2's are the parameters corresponding to visibility at Class I areas in region 2 (Colorado Plateau), if $i \neq 2$, and so forth. Because the model assumes that the relationship between residential visibility and utility is the same everywhere, there is only one θ. The parameter ρ in this CES utility function is an important determinant of the slope of the marginal WTP curve associated with any of the environmental quality variables. When $\rho=1$, the marginal WTP curve is horizontal. When $\rho<1$, it is downward sloping.

The household's budget constraint is:

$$m - p \cdot X \leq 0 ,$$

[39]We remind the reader that, although residential and recreational visibility benefits estimation is discussed simultaneously in this section, benefits are calculated and presented separately for each visibility category.

where m is income, and p is the price of X. Without loss of generality, set p = 1. The only choice variable is X. The household maximizes its utility by choosing X=m. The indirect utility function for a household in the nth residential area and the ith region is therefore

$$V_{ni}(m, Z_n, Q; q, g, d, r) = (m^r + qZ_n^r + \sum_{k=1}^{N_i} g_{ik} Q_{ik}^r + \sum_{j \neq i} \sum_{k=1}^{N_j} d_{jk} Q_{jk}^r)^{1/r} \quad ,$$

where Q denotes the vector of vectors, Q_1, Q_2, Q_3, Q_4, Q_5, and Q_6, and the unsubscripted γ and δ denote vectors as well.

Given estimates of ρ, θ, the γ's and the δ's, the household's utility function and the corresponding WTP functions are fully specified. The household's WTP for any set of changes in the levels of visibility at in-region Class I areas, out-of-region Class I areas, and the household's residential area can be shown to be:

$$WTP_{ni}(\Delta Z, \Delta Q) = m - [m^r + q(Z_{0n}^r - Z_{1n}^r) + \sum_{k=1}^{N_i} g_{ik}(Q_{0ik}^r - Q_{1ik}^r) + \sum_{j \neq i} \sum_{k=1}^{N_j} d_{jk}(Q_{0jk}^r - Q_{1jk}^r)]^{1/r} \quad .$$

The household's WTP for a single visibility improvement will depend on its order in the series of visibility improvements the household is valuing. If it is the first visibility improvement to be valued, the household's WTP for it follows directly from the previous equation. For example, the household's WTP for an improvement in visibility at the first in-region park, from $Q_{i1} = Q_{0i1}$ to $Q_{i1} = Q_{1i1}$, is

$$WTP(\Delta Q_{i1}) = m - [m^r + g_{i1}(Q_{0i1}^r - Q_{1i1}^r)]^{1/r} \quad ,$$

if this is the first (or only) visibility change the household values.

5.1.2 Measure of Visibility: Environmental "Goods" Versus "Bads"

In the above model, Q and Z are environmental "goods." As the level of visibility increases, utility increases. The utility function and the corresponding WTP function both have reasonable properties. The first derivative of the indirect utility function with respect to Q (or Z) is positive; the second derivative is negative. WTP for a change from Q_0 to a higher (improved) level of visibility, Q_1, is therefore a concave function of Q_1, with decreasing marginal WTP.

The measure of visibility that is currently preferred by air quality scientists is the deciview, which increases as visibility *decreases*. Deciview, in effect, is a measure of the *lack* of visibility. As deciviews increase, visibility, and therefore utility, decreases. The deciview, then, is a measure of an environmental "bad." There are many examples of environmental "bads" – all types of pollution are environmental "bads." Utility decreases, for example, as the concentration of particulate matter in the atmosphere increases.

One way to value decreases in environmental bads is to consider the "goods" with which they are associated, and to incorporate those goods into the utility function. In particular, if B denotes an environmental "bad," such that:

$$\frac{\partial V}{\partial B} < 0 \ ,$$

and the environmental "good," Q, is a function of B,

$$Q = F(B) \ ,$$

then the environmental "bad" can be related to utility via the corresponding environmental "good":[40]

$$V = V(m, Q) = V(m, F(B)) \ .$$

The relationship between Q and B, F(B), is an empirical relationship that must be estimated.

There is a potential problem with this approach, however. If the function relating B and Q is not the same everywhere (i.e., if for a given value of B, the value of Q depends on other factors as well), then there can be more than one value of the environmental good corresponding to any given value of the environmental bad, and it is not clear which value to use. This has been identified as a problem with translating deciviews (an environmental "bad") into visual range (an environmental "good"). It has been noted that, for a given deciview value, there can be many different visual ranges, depending on the other factors that affect visual range – such as light angle and altitude. We note here, however, that this problem is not unique to visibility, but is a general problem when trying to translate environmental "bads" into "goods."[41]

In order to translate deciviews (a "bad") into visual range (a "good"), we use a relationship derived by Malm and Pitchford (1994) in which

$$DV = 10 * ln(\frac{391}{VR}) \ ,$$

where DV denotes deciview and VR denotes visual range (in kilometers). Solving for VR as a function of DV yields

---

[40] There may be more than one "good" related to a given environmental "bad." To simplify the discussion, however, we assume only a single "good."

[41] Another example of an environmental "bad" is particulate matter air pollution (PM). The relationship between survival probability (Q) and the ambient PM level is generally taken to be of the form

$$Q = 1 - \alpha e^{\beta PM} .$$

where α denotes the mortality rate (or level) when there is no ambient PM (i.e., when PM=0). However, α is implicitly a function of all the factors other than PM that affect mortality. As these factors change (e.g., from one location to another), α will change (just as visual range changes as light angle changes). It is therefore possible to have many values of Q corresponding to a given value of PM, as the values of α vary.

$$VR = 391 * e^{-0.1DV}.$$

This conversion is based on specific assumptions characterizing the "average" conditions of those factors, such as light angle, that affect visual range. To the extent that specific locations depart from the average conditions, the relationship will be an imperfect approximation.[42]

5.1.3 Estimating the Parameters for Visibility at Class I Areas: the γ's and δ's

As noted in Section 2, if we consider a particular visibility change as the first or the only visibility change valued by the household, the household's WTP for that change in visibility can be calculated, given income (m), the "shape" parameter, ρ, and the corresponding recreational visibility parameter. For example, a Southeast household's WTP for a change in visibility at in-region parks (collectively) from $Q_1 = Q_{01}$ to $Q_1 = Q_{11}$ is:

$$WTP(DQ_1) = m - [m^r + g_1(Q_{01}^r - Q_{11}^r)]^{1/r}$$

if this is the first (or only) visibility change the household values.

Alternatively, if we have estimates of m as well as WTP_1^{in} and WTP_1^{out} of in-region and out-of-region households, respectively, for a given change in visibility from Q_{01} to Q_{11} in Southeast parks, we can solve for γ_1 and δ_1 as a function of our estimates of m, WTP_1^{in} and WTP_1^{out}, for any given value of ρ. Generalizing, we can derive the values of γ and δ for the j^{th} region as follows:

$$g_j = \frac{(m - WTP_j^{in})^r - m^r}{(Q_{0j}^r - Q_{1j}^r)}$$

and

$$d_j = \frac{(m - WTP_j^{out})^r - m^r}{(Q_{0j}^r - Q_{1j}^r)}.$$

Chestnut and Rowe (1990) and Chestnut (1997) estimated WTP (per household) for specific visibility changes at national parks in three regions of the United States – both for households that are in-region (in the same region as the park) and for households that are out-of-region. The Chestnut and Rowe study asked study subjects what they would be willing to pay for each of three visibility improvements in the national parks in a given region. Study subjects were shown a map of the region, with dots indicating the locations of the parks in question. The WTP questions referred to the three visibility improvements in all the parks collectively; the survey did not ask subjects' WTP for these improvements in specific parks individually. Responses were categorized according to whether the respondents lived in the same region as

[42] Ideally, we would want the location- , time-, and meteorological condition-specific relationships between deciviews and visual range, which could be applied as appropriate. This is probably not feasible, however.

the parks in question ("in-region" respondents) or in a different region ("out-of-region" respondents). The areas for which in-region and out-of-region WTP estimates are available from Chestnut and Rowe (1990), and the sources of benefits transfer-based estimates that we employ in the absence of estimates, are summarized in Exhibit 5-1. In all cases, WTP refers to WTP per household.

Exhibit 5-1 Available Information on WTP for Visibility Improvements in National Parks

Region of Park	Region of Household	
	In-Region[a]	**Out-of-Region**[b]
1. California	WTP estimate from study	WTP estimate from study
2. Colorado Plateau	WTP estimate from study	WTP estimate from study
3. Southeast United States	WTP estimate from study	WTP estimate from study
4. Northwest United States	(based on benefits transfer from California)	
5. Northern Rockies	(based on benefits transfer from Colorado Plateau)	
6. Rest of United States	(based on benefits transfer from Southeast U.S.)	

[a] In-region" WTP is WTP for a visibility improvement in a park in the same region as that in which the household is located. For example, in-region WTP in the "Southeast" row is the estimate of the average Southeast household's WTP for a visibility improvement in a Southeast park.

[b] Out-of-region" WTP is WTP for a visibility improvement in a park that is not in the same region in which the household is located. For example, out-of-region WTP in the "Southeast" row is the estimate of WTP for a visibility improvement in a park in the Southeast by a household outside of the Southeast.

In the primary calculation of visibility benefits for this analysis, only visibility changes at parks within visibility regions for which a WTP estimate was available from Chestnut and Rowe (1990) are considered (for both in- and out-of-region benefits). Primary estimates will not include visibility benefits calculated by transferring WTP values to visibility changes at parks not included in the Chestnut and Rowe study. Transferred benefits at parks located outside of the Chestnut and Rowe visibility regions will, however, be included as an alternative calculation.

The values of the parameters in a household's utility function will depend on where the household is located. The region-specific parameters associated with visibility at Class I areas (that is, all parameters except the residential visibility parameter) are arrayed in Exhibit 5-2. The parameters in columns 1-3 can be directly estimated using WTP estimates from Chestnut and Rowe (1990) (the columns labeled "Region 1," "Region 2," and "Region 3").

Exhibit 5-2 Summary of Region-Specific Recreational Visibility Parameters to be Estimated in Household Utility Functions

Region of Household	Region of Park					
	Region 1	Region 2	Region 3	Region 4	Region 5	Region 6
Region 1	γ_1[a]	δ_2	δ_3	δ_4	δ_5	δ_6
Region 2	δ_1	γ_2	δ_3	δ_4	δ_5	δ_6
Region 3	δ_1	δ_2	γ_3	δ_4	δ_5	δ_6
Region 4	δ_1	δ_2	δ_3	γ_4	δ_5	δ_6
Region 5	δ_1	δ_2	δ_3	δ_4	γ_5	δ_6
Region 6	δ_1	δ_2	δ_3	δ_4	δ_5	γ_6

[a] The parameters arrayed in this table are region-specific rather than park-specific or wilderness area-specific. For example, δ_1 is the parameter associated with visibility at " Class I areas in region 1" for a household in any region other than region 1. The benefits analysis must derive Class I area-specific parameters – e.g., δ_{1k}, for the kth Class I area in the first region.

For the three regions covered in Chestnut and Rowe (1990) (California, the Colorado Plateau, and the Southeast United States), we can directly use the in-region WTP estimates from the study to estimate the parameters in the utility functions corresponding to visibility at in-region parks (γ_1); similarly, we can directly use the out-of-region WTP estimates from the study to estimate the parameters for out-of-region parks (δ_1). For the other three regions not covered in the study, however, we must rely on benefits transfer to estimate the necessary parameters.

While Chestnut and Rowe (1990) provide useful information on households' WTP for visibility improvements in national parks, there are several significant gaps remaining between the information provided in that study and the information necessary for the benefits analysis. First, as noted above, the WTP responses were not park-specific, but only region-specific. Because visibility improvements vary from one park in a region to another, the benefits analysis must value park-specific visibility changes. Second, not all Class I areas in each of the three regions considered in the study were included on the maps shown to study subjects. Because the focus of the study was primarily national parks, most Class I wilderness areas were not included. Third, only three regions of the United States were included, leaving the three remaining regions without direct WTP estimates.

In addition, Chestnut and Rowe (1990) elicited WTP responses for *three different* visibility changes, rather than a single change. In theory, if the CES utility function accurately describes household preferences, and if all households in a region have the same preference structure, then households' three WTP responses corresponding to the three different visibility changes should all produce the same value of the associated recreational visibility parameter, given a value of ρ and an income, m. In practice, of course, this is not the case.

In addressing these issues, we take a three-phase approach:

(1) We estimate region-specific parameters for the region in the modeled domain covered by Chestnut and Rowe (1990) (California, the Colorado Plateau, and the Southeast) – γ_1, γ_2, and γ_3 and δ_1, δ_2, and δ_3. **(2)** We infer region-specific parameters for those regions not covered by the Chestnut and Rowe study (the Northwest United States, the Northern Rockies, and the rest of the U.S.) – γ_4, γ_5, and γ_6 and δ_4, δ_5, and δ_6. **(3)** We derive park- and wilderness area-specific parameters within each region (γ_{1k} and δ_{1k}, for k=1, ..., N_1; γ_{2k} and δ_{2k}, for k=1, ..., N_2; and so forth).

The question that must be addressed in the first phase is how to estimate a single region-specific in-region parameter and a single region-specific out-of-region parameter for each of the three regions covered in Chestnut and Rowe (1990) from study respondents' WTPs for *three different* visibility changes in each region. All parks in a region are treated collectively as if they were a single "regional park" in this first phase. In the second phase, we infer region-specific recreational visibility parameters for regions not covered in the Chestnut and Rowe study (the Northwest United States, the Northern Rockies, and the rest of the U.S.). As in the first phase, we ignore the necessity to derive park-specific parameters at this phase. Finally, in the third phase, we derive park- and wilderness area-specific parameters for each region.

Estimating Region-Specific Recreational Visibility Parameters for the Region Covered in the Chestnut and Rowe Study (Regions 1, 2, and 3)

Given a value of ρ and estimates of m and in-region and out-of-region WTPs for a change from Q_0 to Q_1 in a given region, the in-region parameter, γ, and the out-of-region parameter, δ, for that region can be solved for. Chestnut and Rowe (1990), however, considered not just one, but three visibility changes in each region, each of which results in a different calibrated γ and a different calibrated δ, even though in theory all the γ's should be the same and similarly, all the δ's should be the same. For each region, however, we must have only a single γ and a single δ.

Denoting $\hat{\gamma}_j$ as our estimate of γ for the j^{th} region, based on all three visibility changes, we chose $\hat{\gamma}_j$ to best predict the three WTPs observed in the study for the three visibility improvements in the j^{th} region. First, we calculated $\hat{\gamma}_{ji}$, i=1, 2, 3, corresponding to each of the three visibility improvements considered in the study. Then, using a grid search method beginning at the average of the three $\hat{\gamma}_{ji}$'s, we chose $\hat{\gamma}_j$ to minimize the sum of the squared differences between the WTPs we predict using $\hat{\gamma}_j$ and the three region-specific WTPs observed in the study. That is, we selected $\hat{\gamma}_j$ to minimize:

$$\sum_{i=1}^{3} (WTP_{ij}(\hat{g}_j) - WTP_{ij})^2$$

where WTP_{ij} and $WTP_{ij}(\hat{\gamma}_j)$ are the observed and the predicted WTPs for a change in visibility in the j^{th} region from $Q_0 = Q_{0i}$ to $Q_1 = Q_{1i}$, i=1, ..., 3. An analogous procedure was used to select an optimal δ, for each of the three regions in the Chestnut and Rowe study.

Inferring Region-Specific Recreational Visibility Parameters for Regions Not Covered in the Chestnut and Rowe Study (Regions 4, 5, and 6)

One possible approach to estimating region-specific parameters for regions not covered by Chestnut and Rowe (1990) (γ_4, γ_5, and γ_6 and δ_4, δ_5, and δ_6) is to simply assume that households' utility functions are the same everywhere, and that the environmental goods being valued are the same – e.g., that a change in visibility at national parks in California is the same environmental good to a Californian as a change in visibility at national parks in Minnesota is to a Minnesotan.

For example, to estimate δ_4 in the utility function of a California household, corresponding to visibility at national parks in the Northwest United States, we might assume that out-of-region WTP for a given visibility change at national parks in the Northwest United States is the same as out-of-region WTP for the same visibility change at national parks in California (income held constant). Suppose, for example, that we have an estimated mean WTP of out-of-region households for a visibility change from Q_{01} to Q_{11} at national parks in Califonia (region 1), denoted WTP_1^{out}. Suppose the mean income of the out-of-region subjects in the study was m. We might assume that, for the same change in visibility at national parks in the Northwest United States, $WTP_4^{out} = WTP_1^{out}$ among out-of-region individuals with income m.

We could then derive the value of δ_4, given a value of ρ as follows:

$$d_4 = \frac{(m - WTP_4^{out})^r - m^r}{Q_{04}^r - Q_{14}^r}$$

where $Q_{04} = Q_{01}$ and $Q_{14} = Q_{11}$, (i.e., where it is *the same* visibility change in parks in region 4 that was valued at parks in the region 1).

This benefits transfer method assumes that (1) all households have the same preference structures and (2) what is being valued in the Northwest United States (by a California household) is the same as what is being valued in the California (by all out-of-region households). While we cannot know the extent to which the first assumption approximates reality, the second assumption is clearly problematic. National parks in one region are likely to differ from national parks in another region in both quality and quantity (i.e., number of parks).

One statistic which is likely to reflect both the quality and quantity of national parks in a region is the average annual visitation rate to the parks in that region. A reasonable way to gauge the extent to which out-of-region people would be willing to pay for visibility changes in parks in the Northwest United States versus in California might be to compare visitation rates in the two regions.[43] Suppose, for example, that twice as many visitor-days are spent in California parks per year as in parks in the Northwest United States per year. This could be an indication that the parks in California are in some way more desirable than those in the Northwest United States and/or that there are more of them -- i.e., that the environmental goods being valued in the two regions ("visibility at national parks") are not the same.

A preferable way to estimate δ_4, then, might be to assume the following relationship:

$$\frac{WTP_4^{out}}{WTP_1^{out}} = \frac{n_4}{n_1}$$

[43] We acknowledge that reliance on visitation rates does not get at nonuse value.

(income held constant), where n_1 = the average annual number of visitor-days to California parks and n_4 = the average annual number of visitor-days to parks in the Northwest United States. This implies that

$$WTP_4^{out} = \frac{n_4}{n_1} * WTP_1^{out}$$

for the same change in visibility in region 4 parks among out-of-region individuals with income m. If, for example, $n_1 = 2n_4$, WTP_4^{out} would be half of WTP_1^{out}. The interpretation would be the following: California national parks have twice as many visitor-days per year as national parks in the Northwest United States; therefore they must be twice as desirable/plentiful; therefore, out-of-region people would be willing to pay twice as much for visibility changes in California parks as in parks in the Northwest United States; therefore a Californian would be willing to pay only half as much for a visibility change in national parks in the Northwest United States as an out-of-region individual would be willing to pay for the same visibility change in national parks in California. This adjustment, then, is based on the premise that the environmental goods being valued (by people out-of-region) are not the same in all regions.

The parameter δ_4 is estimated as shown above, using this adjusted WTP_4^{out}. The same procedure is used to estimate δ_5 and δ_6. We estimate γ_4, γ_5, and γ_6 in an analogous way, using the in-region WTP estimates from the transfer regions, e.g.,

$$WTP_4^{in} = \frac{n_4}{n_1} * WTP_1^{in} \quad .$$

Estimating Park- and Wilderness Area-Specific Parameters

As noted above, Chestnut and Rowe (1990) estimated WTP for a region's national parks collectively, rather than providing park-specific WTP estimates. The γ's and δ's are therefore the parameters that would be in household utility functions if there were only a single park in each region, or if the many parks in a region were effectively indistinguishable from one another. Also noted above is the fact that the Chestnut and Rowe study did not include all Class I areas in the regions it covered, focusing primarily on national parks rather than wilderness areas. Most Class I wilderness areas were not represented on the maps shown to study subjects. In California, for example, there are 31 Class I areas, including 6 national parks and 25 wilderness areas. The Chestnut and Rowe study map of California included only 10 of these Class I areas, including all six of the national parks. It is unclear whether subjects had in mind "all parks and wilderness areas" when they offered their WTPs for visibility improvements, or whether they had in mind the specific number of (mostly) parks that were shown on the maps. The derivation of park- and wilderness area-specific parameters depends on this.

Derivation of Region-specific WTP for National Parks and Wilderness Areas

If study subjects were lumping all Class I areas together in their minds when giving their WTP responses, then it would be reasonable to allocate that WTP among the specific parks and wilderness areas in the region to derive park- and wilderness area-specific γ's and δ's for the region. If, on the other hand, study subjects were thinking only of the (mostly) parks shown on the map when they gave their WTP

response, then there are two possible approaches that could be taken. One approach assumes that households would be willing to pay some additional amount for the same visibility improvement in additional Class I areas that were not shown, and that this additional amount can be estimated using the same benefits transfer approach used to estimate region-specific WTPs in regions not covered by Chestnut and Rowe (1990).

However, even if we believe that households would be willing to pay some additional amount for the same visibility improvement in additional Class I areas that were not shown, it is open to question whether this additional amount can be estimated using benefits transfer methods. A third possibility, then, is to simply omit wilderness areas from the benefits analysis. For this analysis we calculate visibility benefits assuming that study subjects lumped all Class I areas together when stating their WTP, even if these Class I areas were not present on the map.

Derivation of park- and wilderness area-specific WTPs, given region-specific WTPs for national parks and wilderness areas

The first step in deriving park- and wilderness area-specific parameters is the estimation of park- and wilderness area-specific WTPs. To derive park and wilderness area-specific WTPs, we apportion the region-specific WTP to the specific Class I areas in the region according to each area's share of the region's visitor-days. For example, if WTP_1^{in} and WTP_1^{out} denote the mean household WTPs in the Chestnut and Rowe (1990) study among respondents who were in-region-1 and out-of-region-1, respectively, n_{1k} denotes the annual average number of visitor-days to the kth Class I area in California, and n_1 denotes the annual average number of visitor-days to all Class I areas in California (that are included in the benefits analysis), then we assume that

$$WTP_{1k}^{in} = \frac{n_{1k}}{n_1} * WTP_1^{in} \ ,$$

and

$$WTP_{1k}^{out} = \frac{n_{1k}}{n_1} * WTP_1^{out}.$$

Using WTP_j^{in} and WTP_j^{out}, either from the Chestnut and Rowe study (for j = 1, 2, and 3) or derived by the benefits transfer method (for j = 4, 5, and 6), the same method is used to derive Class I area-specific WTPs in each of the six regions.

While this is not a perfect allocation scheme, it is a reasonable scheme, given the limitations of data. Visitors to national parks in the United States are not all from the United States, and certainly not all from the region in which the park is located. A very large proportion of the visitors to Yosemite National Park in California, for example, may come from outside the U.S. The above allocation scheme implicitly assumes that the relative frequencies of visits to the parks in a region *from everyone in the world* is a

reasonable index of the relative WTP of an average household in that region (WTP_j^{in}) or out of that region (but in the U.S.) (WTP_j^{out}) for visibility improvements at these parks.[44]

A possible problem with this allocation scheme is that the relative frequency of visits is an indicator of use value but not necessarily of nonuse value, which may be a substantial component of the household's total WTP for a visibility improvement at Class I areas. If park A is twice as popular (i.e., has twice as many visitors per year) as park B, this does not necessarily imply that a household's WTP for an improvement in visibility at park A is twice its WTP for the same improvement at park B. Although an allocation scheme based on relative visitation frequencies has some obvious problems, however, it is still probably the best way to allocate a collective WTP.

Derivation of park- and wilderness area-specific parameters, given park- and wilderness area-specific WTPs

Once the Class I area-specific WTPs have been estimated, we could derive the park- and wilderness area-specific γ's and δ's using the method used to derive region-specific γ's and δ's. Recall that that method involved (1) calibrating γ and δ to each of the three visibility improvements in the Chestnut and Rowe study (producing three γ's and three δ's), (2) averaging the three γ's and averaging the three δ's, and finally, (3) using these average γ and δ as starting points for a grid search to find the optimal γ and the optimal δ – i.e., the γ and δ that would allow us to reproduce, as closely as possible, the three in-region and three out-of-region WTPs in the study for the three visibility changes being valued.

Going through this procedure for each national park and each wilderness area separately would be very time consuming, however. We therefore used a simpler approach, which produces very close approximations to the γ's and δ's produced using the above approach. If:

WTP_j^{in} =	the in-region WTP for the change in visibility from Q_0 to Q_1 in the jth region;
WTP_{jk}^{in}=	the in-region WTP for the same visibility change (from Q_0 to Q_1) in the kth Class I area in the jth region (= s_{jk}*WTP_j^{in}, where s_{jk} is the kth area's share of visitor-days in the jth region);
m =	income;
γ_j* =	the optimal value of γ for the jth region; and
γ_{jk} =	the value of γ_{jk} calibrated to WTP_{jk}^{in} and the change from Q_0 to Q_1;

then[45]:

[44] This might be thought of as two assumptions: (1) that the relative frequencies of visits to the parks in a region *from everyone in the world* is a reasonable representation of the relative frequency of visits *from people in the United States* – i.e., that the parks that are most popular (receive the most visitors per year) in general are also the most popular among Americans; and (2) that the relative frequency with which Americans visit each of their parks is a good index of their relative WTPs for visibility improvements at these parks.

[45] γ_j* is only approximately equal to the right-hand side because, although it is the optimal value designed to reproduce as closely as possible all three of the WTPs corresponding to the three visibility changes in the Chestnut and Rowe study, γ_j* will not exactly reproduce any of these WTPs.

$$\gamma_j^* \approx \frac{(m - WTP_j^{in})^r - m^r}{(Q_0^r - Q_1^r)}$$

and

$$\gamma_{jk} = \frac{(m - WTP_{jk}^{in})^r - m^r}{(Q_0^r - Q_1^r)}$$

which implies that:

$$\gamma_{jk} \approx \alpha_{jk} * \gamma_j^* ,$$

where:

$$\alpha_{jk} = \frac{(m - WTP_{jk}^{in})^r - m^r}{(m - WTP_j^{in})^r - m^r} .$$

We use the adjustment factor, a_{jk}, to derive γ_{jk} from γ_j*, for the kth Class I area in the jth region. We use an analogous procedure to derive δ_{jk} from δ_j* for the kth Class I area in the jth region (where, in this case, we use WTP_j^{out} and WTP_{jk}^{out} instead of WTP_j^{in} and WTP_{jk}^{in}).[46]

5.1.4 Estimating the Parameter for Visibility in Residential Areas: θ

The estimate of θ is based on McClelland et al. (1991), in which household WTP for improvements in residential visibility was elicited from respondents in Chicago and Atlanta. A notable difference between the Chestnut and Rowe study and the McClelland study is that, while the former elicited WTP responses for three different visibility changes, the latter considered only one visibility change. The estimation of θ was therefore a much simpler procedure, involving a straightforward calibration to the single income and WTP in the study:

$$\theta = \frac{(m - WTP)^r - m^r}{(Z_0^r - Z_1^r)} .$$

5.1.5 Putting it All Together: the Household Utility and WTP Functions

[46] This method uses a single in-region WTP and a single out-of-region WTP per region. Although the choice of WTP will affect the resulting adjustment factors (the a_{jk}'s) and therefore the resulting γ_{jk}'s and δ_{jk}'s, the effect is negligible. We confirmed this by using each of the three in-region WTPs in California and comparing the resulting three sets of γ_{jk}'s and δ_{jk}'s, which were different from each other by about one one-hundredth of a percent.

Given an estimate of θ, derived as shown in Section 5, and estimates of the γ's and δ's, derived as shown in Section 4, based on an assumed or estimated value of ρ, the utility and WTP functions for a household in any region are fully specified. We can therefore estimate the value to that household of visibility changes from any baseline level to any alternative level in the household's residential area and/or at any or all of the Class I areas in the United States, in a way that is consistent with economic theory. In particular, the WTP of a household in the ith region and the nth residential area for any set of changes in the levels of visibility at in-region Class I areas, out-of-region Class I areas, and the household's residential area (given by equation (24)) is:

$$WTP_{ni}(\Delta Z,\Delta Q) = m - [m^r + q(Z_{0n}^r - Z_{1n}^r) + \sum_{k=1}^{N_i} g_{ik}(Q_{0ik}^r - Q_{1ik}^r) + \sum_{j\neq i}\sum_{k=1}^{N_j} d_{jk}(Q_{0jk}^r - Q_{1jk}^r)]^{1/r} \quad .$$

The national benefits associated with any suite of visibility changes is properly calculated as the sum of these household WTPs for those changes. The benefit of any subset of visibility changes (e.g., changes in visibility only at Class I areas in California) can be calculated by setting all the other components of the WTP function to zero (that is, by assuming that all other visibility changes that are not of interest are zero). This is effectively the same as assuming that the subset of visibility changes of interest is the first or the only set of changes being valued by households. Estimating benefit components in this way will yield slightly upward biased estimates of benefits, because disposable income, m, is not being reduced by the WTPs for any prior visibility improvements. That is, each visibility improvement (e.g., visibility at Class I areas in the California) is assumed to be the first, and they cannot all be the first. The upward bias should be extremely small, however, because all of the WTPs for visibility changes are likely to be very small relative to income.

5.2 AGRICULTURAL BENEFITS

Changes in ozone concentrations are known to affect agricultural production, affecting agricultural crops to different degrees depending on their sensitivity. Estimating the economic benefits associated with these changes in production requires several steps. Estimated changes in ozone concentrations are combined with experimental dose-response functions to estimate crop yield changes. The effect of yield changes on agricultural cropping decisions and resulting production and prices are then evaluated using a model of the agricultural sector, resulting in estimates of changes in farm income and consumer welfare. Each of the steps involved in this analysis is described in more detail in the following sections. Section 5.2.1 describes the source of exposure-response functions and the selection of an index of ozone exposure. Section 5.2.2 describes the derivation of estimated ozone concentrations under alternative regulatory profiles. The method for estimating yield changes is described in Section 5.2.3, and the agricultural model used to estimate the impact of changes in yield is discussed in Section 5.2.4. The results are presented in Chapter 6.

5.2.1 Exposure-Response Functions

Experimental data to evaluate the response of crops to ozone has been collected for a limited number of crops under the National Crop Loss Assessment Network (NCLAN) program. The objective of this program was to employ a consistent experimental methodology to provide comparable results across crops. The crops included in the NCLAN experiments are corn, cotton, peanuts, sorghum, soybeans, winter wheat, potatoes, lettuce, kidney beans, tomatoes, and hay. For many crops, the NCLAN program evaluated the effects of ozone on several different cultivars. Although not necessarily representative of the full range of variability in crop response, the results for different cultivars do permit identification of a

range of responsiveness. The most tolerant and responsive functions are used to represent minimum and maximum impacts, *within the limits of available data*.

In its analysis of the welfare benefits associated with ozone National Ambient Air Quality Standards (NAAQS), U.S. EPA elected to represent crop exposure to ozone as a cumulative index (U.S. EPA, 1996b). The index selected is the SUM06 index, which sums the ozone concentration for every hour that exceeds 0.06 ppm, within a 12-hour period from 8:00 A.M. to 8:00 P.M.

Use of cumulative exposure-response functions is relatively recent, and few experiments have been designed or reported in terms of the SUM06 index. Because the NCLAN program used a consistent protocol and developed a database of experimental conditions and results for all of its studies, U.S. EPA's Environmental Research Laboratory (ERL) was able to use original data from NCLAN studies to develop SUM06 exposure response functions for most NCLAN crops[47] (Lee and Hogsett, 1996). In addition, the agricultural model used in this analysis does not reflect non-commodity crops such as lettuce and kidney beans (described below). Exhibit 5-3 presents the exposure-response functions used in this analysis.

Exhibit 5-3 Ozone Exposure-Response Functions for Selected Crops (SUM06)

Ozone Index	Quantity	Crop	Function	Median Experimental Duration (Days)	Median Duration (Months)
SUM06	Max	Cotton	1-exp(-(index/78)^1.311)	119	4
SUM06	Max	Field Corn	1-exp(-(index/92.4)^2.816)	83	3
SUM06	Max	Grain Sorghum	1-exp(-(index/177.8)^2.329)	85	3
SUM06	Max	Peanut	1-exp(-(index/99.8)^2.219)	112	4
SUM06	Max	Soybean	1-exp(-(index/131.4)^1)	104	3
SUM06	Max	Winter Wheat	1-exp(-(index/27.2)^1.0)	58	2
SUM06	Min	Cotton	1-exp(-(index/116.8)^1.523)	119	4
SUM06	Min	Field Corn	1-exp(-(index/94.2)^4.307)	83	3
SUM06	Min	Grain Sorghum	1-exp(-(index/177.8)^2.329)	85	3
SUM06	Min	Peanut	1-exp(-(index/99.8)^2.219)	112	4
SUM06	Min	Soybean	1-exp(-(index/299.7)^1.547)	104	3
SUM06	Min	Winter Wheat	1-exp(-(index/72.1)^2.353)	58	2

Source: Lee and Hogsett (1996)

[47]Data were not sufficient to develop functions for tomatoes or hay.

The form of these functions is a Weibull specification transformed to predict a yield loss relative to conditions of "clean air", or a zero SUM06 value. The resulting equation is in the form of:

$$Y = 1 - e^{[-(SUM06/B)^{\wedge}C]},$$

where:

Y	=	predicted relative yield loss (PRYL), expressed as a decimal value (i.e., not multiplied by 100 to report as a percent loss), and relative to a zero SUM06 (or clean air) condition
SUM06	=	cumulative SUM06 ozone statistic at a specified level of spatial representation, in ppm
B, C	=	statistically estimated parameters, unitless.

Application of Exposure Response Functions to a Non-Zero Baseline

There is an issue associated with applying the yield loss functions to analysis of alternative regulatory profiles. The functions provide a predicted yield loss relative to "clean" air, while regulatory analysis needs to compare regulatory options to a baseline, non-zero ozone condition. Therefore, the yield change resulting from the regulatory scenario is evaluated as the yield loss relative to clean air under the regulatory scenario being evaluated compared to the yield loss under baseline conditions.

To address this issue, the change in yield under clean air conditions can be divided by the baseline yield. If yield under clean conditions is 100 percent of possible yield, then baseline yield in this context is 1 minus baseline yield loss. Thus the change in yields relative to the baseline can be given as:

$$(PRYL_{baseline} - PRYL_{control})/(1-PRYL_{baseline}).$$

Ozone Index Computation

In order to accurately reflect changes in yields using exposure response functions, they must be applied in a way that is consistent with the experimental conditions used to generate the functions. Specifically, the ozone index, in this case the SUM06 index, needs to be consistent with ozone exposure used in the experimental derivation of the function. For example, if the function is a 12-hour exposure function, then the index used must be a 12-hour index. Another component of the experimental exposure is the duration of the experiment. A precise reflection of experimental conditions would require that the ozone index should be calculated for the same number of days as used in the experiment for each crop. However, in the benefits analysis for the 1997 ozone NAAQS RIA, it was determined that the median duration of all NCLAN experiments for a given crop provided a statistically sound reflection of duration for the purposes of estimating SUM06 indices for estimating agricultural benefits (Mathtech1997). The median durations for each crop are reported in Exhibit 5-3 in both days and months. The ozone NAAQS analysis constructed the ozone index based on the nearest number of months; this analysis constructed the index based on the number of days.

Finally, because growing seasons vary throughout the U.S., the exposure needs to reflect the months in which a crop would be grown in a given location. To calculate the SUM06 index for the

appropriate growing season, state-level data on planting and harvesting dates was used in this analysis[48] (U.S. Department of Agriculture, 1984; U.S. EPA, 1993). To calculate the cumulative SUM06 index, the experimental duration for each crop was anchored on that crop's harvest date in each state in order to most closely approximate the relevant period of exposure for yield analysis. The harvest date was assumed to be the first day in the month of harvest, so that the SUM06 index includes the months up to but not including the harvest month.

The baseline and control ozone data for this analysis were developed from monthly SUM06 values, requiring several steps in the calculation of a duration-based index. First, starting at the month before the harvest month, each full month of SUM06 data was summed. The ozone value for the first month of the duration period was calculated as the fraction of the remaining days in the duration period to the number of days in the month. For example, soybeans have a 104-day duration, translating to 3 full months plus a fraction of the first month in the growing season. If soybeans are harvested in October in a given state, three full months of data starting in September are summed (91 days), along with 13 days of June, or 0.43 of the June SUM06 data, to obtain the 104-day SUM06 index. This approach implicitly assumes an equal average daily SUM06 within each bi-monthly period. The index was calculated on a county level assuming all counties reflect the state-level growing seasons.

While the ozone data in this analysis were modeled from May through September, the growing season for some crops includes April, October, and November. To estimate SUM06 values for these unmodeled months, base-year ozone values were used. For the Western U.S., the available data were historical monitor-level ozone values for 1995, and for the Eastern U.S., we used data from 1996.

5.2.2 Estimation of Yield Changes

In this analysis, use of a single exposure response function to estimate changes in yields implies that all producers are using a single cultivar of a given crop. This, combined with the limited number of cultivars evaluated in the NCLAN program, introduces an unquantifiable uncertainty into the estimation of yield changes. The most sensitive cultivar was used to represent the upper bound of the range that could be estimated, and the least sensitive cultivar was used to represent the lower bound of that range.

Using the exposure response functions and the SUM06 ozone indices, county-level yield changes were estimated between each regulatory profile and the baseline. County level yield changes were then aggregated to the state level using 1997 data on county level production as weights (U.S. Department of Agriculture, 1988a): the resulting state-level yield changes were used for quality control purposes. The model used to estimate changes in the agricultural sector resulting from yield changes (described in Section 3.4, below) requires a national level yield change; this was calculated in the same manner as was the change in state-level yields.

5.2.3 AGSIM© MODEL

AGSIM© is an econometric-simulation model that is based on a large set of statistically estimated demand and supply equations for agricultural commodities produced in the United States. This model has

[48]Peanut emergence and harvest dates were taken from the U.S. EPA PRZM-2 Model data.

been peer-reviewed and utilized in many pesticide and other major agricultural policy evaluations (Taylor et al., 1993).

The model is capable of analyzing the effects of changes in policies that affect crop yields or production costs. This is achieved by estimating how farmers will adjust crop acreage between commodities when relative profitability changes as a result of policy-induced crop yield and/or production cost changes. Acreage and yield changes from various scenarios will affect total production of crops, which simultaneously affects both commodity prices and consumption. Commodity price changes, in turn, affect profitability and cropping patterns in subsequent years. Federal farm program and conservation reserve effects are also incorporated into the model. The model has been adapted to reflect the projections to 2010 from the last future year for which baseline forecasts are available: 2007. Although ozone impacts will be experienced far in the future, it was not possible to forecast the AGSIM© model far beyond USDA baseline forecasts that extend to 2007. Therefore, the 2030 ozone conditions were modeled using the 2010 version of the model.

Model Specification

AGSIM© is based on a set of dynamic supply and demand equations for major crops. Commodities are generally linked on both the supply side and demand side of markets. Crops included in the model are corn, grain sorghum, barley, oats, wheat, soybeans, cotton, hay, peanuts and rice. The simulation component of the model finds the set of prices for all commodities endogenous to the model that simultaneously clear all markets in each year over the simulation period. Dynamics are incorporated into the econometric specification and thus incorporated into the simulation model. All equations in the model were econometrically estimated, except a few policy equations that were based on legislated formula.

Supply Components

The crop supply component of AGSIM© is based on a set of supply equations for the major field crops produced in the United States. Effects of farm programs, specifically the 1985 Food Security Act (FSA), the 1990 Food Agricultural Conservation and Trade Act (FACTA), and the 1996 Federal Agricultural Improvement and Reform Act (FAIR), are reflected in the econometric specification of the supply component of the model, and thus are included in the simulation model.

Ex ante simulation of environmental policy will likely involve an assumption of continuation of the 1996 FAIR Act indefinitely. However, since most of the historical observations on which supply equations were econometrically estimated occurred under different programs, it is important to consider how historical equations reflect the 1996 FAIR Act. The basic philosophy that guided inclusion of farm program features into the supply component of the model follow. First, beginning with the 1985 FSA, continuing with the 1990 FACTA, and now with the 1996 FAIR Act, North American Free Trade Agreement (NAFTA) and the General Agreement on Tariffs and Trade (GATT), farm and international trade policy has moved U.S. agriculture to a market orientation. Although the 1985 FSA and the 1990 FACTA had price support and acreage diversion features, they embodied a strong market orientation. For all major program crops (in AGSIM©), the acreage devoted to the crop exceeded the acreage under government programs. Thus, *at the margin*, market prices (and not support prices) influenced crop acreage. Another way of looking at this is that farm programs have influenced crops at the intra-margin, while the market has influenced crops at the margin. Thus, after accounting for acreage diverted under

farm programs, expected prices determine acreage. For these reasons, AGSIM© should be valid for simulating agricultural markets under the market conditions established under the 1996 FAIR Act.

Sets of equations that comprise the supply component of the current version of the model include: (1) acreage planted to each crop, (2) acreage harvested of each crop, (3) acreage in annual set-aside or acreage reduction programs (ARP) by crop, (4) acreage in cultivated summer fallow, (5) crop yields per harvested acre, (6) rate of participation in Federal farm programs by crop, and (7) annual set-aside rates by crop under past farm programs, as related to stock levels (historically legislated) and thus related to market price. Identities in the model are: (a) production is the product of acreage harvested and yield per harvested acre, and (b) the quantity supplied equals the quantity demanded for each commodity (market clearing). Specification of each of these sets of equations follows.

Acreage Planted Equations. Acreage planted is the key behavioral relationship in the supply component of the model. Acreage planted of a particular crop depends on expected per-acre net returns for that crop, expected per-acre net returns for competing crops, and farm program variables. In algebraic (and Fortran) form, the acreage planted equation is:

(1) acresp(ic,it,irun) = bc(ic) + bap(ic)*acresp(ic,it-1,irun) + bcrp(ic)*acrp(ic,it,irun) + bdiv(ic)*acrediv + brm(ic)*rerntm(ic,it,irun) + ber(ic)*rerentnp(it,irun) + byr(ic)*time(it) + bd83(ic)*dumb83(it)

where:

acresp(ic,it,irun)	=	acreage planted to the ic^{th} crop in the it^{th} year and in simulation "irun",
acrp(ic,it,irun)	=	acreage of crop "ic" that was placed in the conservation reserve program,
acrediv	=	acreage diverted under annual set-aside programs,
rerentm(ic,it,irun)	=	real expected per acre returns over variable costs for the ic^{th} crop,
rerentnp(it,irun)	=	real expected per acre returns over variables costs computed as a weighted average[49] of rerentm(ic,it,irun) over all endogenous crops,
time(it)	=	a time-trend variable, and
dumb83(it)	=	a binary dummy variable to account for the PIK program in crop year 1983.

The remaining variables in equation (1) represent estimated coefficients. A single run of AGSIM involves two simulations, one for the baseline (irun=0) and one for the policy scenario (irun=1). These two simulations are then compared to estimate the economic impacts of the policy scenario.

Expected returns over variable costs, rerentm(ic,it,irun), is defined as:

(1a) rerntm(ic,it,irun) = rp(ic,it-1,irun)*ey(ic,it,irun) - rcost(ic,it,irun)

where:

[49]Weights used in computing a composite expected return variable were the acreage harvested of each crop the previous year divided by total acreage harvested the previous year.

rp(ic,it-1,irun) = real price the previous crop year (actual or simulated, depending on the time period),
ey(ic,it,irun) = expected crop yield, and
rcost(ic,it,irun) = real variable production cost.

Expected yield is based on trend-line regressions:

(1b) ey(ic,it,irun) = [cint(ic) + by(ic)*time(it)]

where:
cint(ic) and by(ic) are estimated coefficients.

In the policy run, expected yield is adjusted for exogenously specified percentage yield changes ("dyld"):

(1c) ey(ic,it,irun) = [cint(ic) + by(ic)*time(it)]*(1.0 + dyld(ic,it)/100.)

Changes in real variable costs of production can also be exogenously specified for the policy simulation run. Thus, yield and cost changes directly impact acreage planted through equation (1), and indirectly impact acreage planted because of the resulting impact on prices in equation (1a) and thus in equation (1).

Given signs and magnitudes of estimated coefficients in equation (1), an increase in expected returns of the ic^{th} crop will increase acreage planted of that crop, while an increase in expected returns of other endogenous crops will decrease acreage of the ic^{th} crop. The estimated coefficient on lagged acreage planted in equation (1) is positive and less than one in value for all crops, which means that acreage planted is dynamically stable. The estimated coefficient on the set-aside acreage is negative and less than one in absolute value for all crops except oats, which reflects acreage slippage in the ARP program. Oats were typically planted to set-aside acreage, thus the estimated coefficient on set-aside acreage is positive in the oats equation, as expected. Further comments will be made on the acreage diverted effects on planted acreage after participation rate and acreage diverted equations, which are endogenous, are presented below.

Acreage Harvested Equations. Acreage harvested depends primarily on acreage planted:

(2) acresh(ic,it,irun) = bch(ic) + baph(ic)*acresp(ic,it,irun) + byrh(ic)*time(it) + bdvh(ic)*acrediv

where:
acresh(ic,it,irun) = the acreage harvested of the ic^{th} crop in the it^{th} year and in simulation "irun",

and other variables are as defined previously.

The estimated coefficient baph(ic) is positive and less than one, indicating that not all planted acreage is harvested, as expected. The coefficient bdvh(ic) on the acreage diverted variable is non-zero for oats only, in which case it is negative. This adjusts oat acreage harvested for the complexity of oats being planted (but not harvested) on ARP acreage. A time-trend variable for corn and grain sorghum, but not other crops shows how harvested acreage as a percentage of planted acreage has been increasing slightly over time.

Participation Rate in Farm Programs. Participation rates in the annual set-aside programs under the 1985 FSA and the 1990 FACTA were endogenized in the model with the set of equations:

(3) part(ic,it,irun) = bcp(ic) + brmp(ic)*rerntm(ic,it,irun) + brpp(ic)*rerntp(ic,it,irun) + byr(ic)*time(ic) + bpart(ic)*part(ic,it-1,irun) + bedpp(ic)*redp(ic,it,irun) + bd83p(ic)*dumb83(it)

where:

part(ic,it,irun) = the participation rate in the farm program for the ic^{th} crop in the it^{th} year and in simulation "irun",

rerntp(ic,it,irun)= real expected returns over variable costs based on the support (target) price for that crop,

redp(ic,it,irun) = real effective acreage diversion payment rate,

and other variables are as defined previously.

Estimated coefficients brpp(ic) are non-negative, indicating that an increase in expected returns based on support price will increase participation, while estimated coefficients brmp(ic) are non-positive, indicating that an increase in expected returns based on expected market price will decrease participation. Lagged participation rate in equation (3) shows strong dynamics with respect to farm program participation.

Acreage Diverted under Farm Programs. Acreage diverted under annual set-aside (or ARP) programs is modeled as:

(4) adiv(ic,it,irun) = bcd(ic) + bd83d(ic)*dumb83(it) + bedpd(ic)*redp(ic,it,irun) + byrd(ic)*time(it) + bpsa(ic)*sa(ic,it,irun)*part(ic,it,irun)

where:

adiv(ic,it,irun) = acreage diverted under annual diversion programs for the ic^{th} crop in the it^{th} year and in simulation "irun",

sa(ic,it,irun) = the set-aside rate specified by the Secretary of Agriculture under 1985 FSA and 1990 FACTA,

and other variables are as defined previously.

Acreage slippage (with respect to historical set-aside) in farm programs is implicit in the model specification, and results from the complex simultaneity of farm program variables in sets of equations (1), (3), and (4).

Acreage in Cultivated Summer Fallow. Acreage in cultivated summer fallow is modeled by the equation:

(5) afl(it,irun) = bcfl + bafl*afl(it-1,irun) + berfl*rerentnp(it,irun) + byrfl*time(it) + bd83fl*dumb83(it)

where:

afl(it,irun) = acreage fallowed in year it in simulation run "irun".

Although the acreage in cultivated summer fallow is highly inelastic, this equation shows that an increase in expected returns based on expected market price results in a small decrease in acreage fallowed.

Demand Components

The crop demand component of AGSIM© is based on a set of demand equations for each crop for utilization categories of (a) imports, (b) exports,(c) livestock feed, (d) food, fiber, ethanol production and other domestic uses, (e) ending stocks, and (f) residual use. Each demand component depends on current market price for that commodity and, where relevant, prices of other commodities. The model specification of each utilization category follows.

Imports. Imports of agricultural commodities are modeled by the set of equations:

(6) qd(ic,it,irun,1) = bim(1,ic) + bim(2,ic)*rp(ic,it,irun)*xrate(ic,it-1,irun) + bim(3,ic)*qd(ic,it-1,irun,1) + bim(4,ic)*time(it) + bim(5,ic)*uspop(it,irun)

where:

qd(ic,it,irun,1)	=	the quantity of crop ic imported in year it in simulation run "irun",
rp(ic,it,irun)	=	real market price,
xrate(ic,it-1,irun)	=	the real trade-weighted exchange rate,
uspop(it,irun)	=	the United States population,

and bim(j,ic) are estimated coefficients. Lagged imports in equation (6) reflects dynamic adjustments.

Exports. Exports of agricultural commodities are modeled by the set of equations:

(7) qd(ic,it,irun,2) = bex(1,ic) + bex(2,ic)*rp(ic,it,irun)*xrate(ic,it-1,irun) + bex(3,ic)* qd(ic,it-1,irun,2) + bex(4,ic)*time(it) + bex(5,ic)*wpop(it,irun)

where:

qd(ic,it,irun,2)	=	the quantity of crop ic exported in year it in simulation run "irun", and
wpop(it,irun)	=	world population.

Feed, Fiber and Crushing Use. Domestic utilization of crops for feed, fiber or crushing (depending on the crop) is modeled by the set of equations:

(8) qd(ic,it,irun,3) = bfd(1,ic) + $\sum_{jc}$bfdcross(ic,jc)*rp(jc,it,irun) + bfd(2,ic)*qd(ic,it-1,irun,3) + bfd(3,ic)*time(it)

where:

qd(ic,it,irun,3)	=	utilization for feed, fiber or crushing.

Note that cross-price effects are incorporated into this set of equations through the set of estimated coefficients bfdcross(ic,jc). Symmetry of cross-price effects, consistent with microeconomic theory, was imposed on estimation so that bfdcross(ic,jc) = bfdcross(jc,ic) for ic $\neq$ jc. Own-price effects are all negative, as expected.

Domestic Food Use. The set of equations to represent domestic food use is:

(9) qd(ic,it,irun,4) = bfo(1,ic) + bfo(2,ic)*rp(ic,it,irun) + bfo(3,ic)*qd(ic,it-1,irun,4) + bfo(4,ic)*time(it) + bfo(5,ic)*uspop(it,irun) + bfo(6,ic)*rdincome(it,irun)

where:

rdincome(it,irun) = real per-capita disposable income in the United States,

and other variables are as defined previously. In the case of peanuts, the real market price is replaced by the fixed quota price that applies to all domestically consumed peanuts. This quota price for peanuts applies to the 1985 FSA, the 1990 FACTA, and continues with the 1996 FAIR Act.

Ending Stocks. Ending stocks are viewed as another component of demand. Although commodities are often held to maintain pipeline inventories, commodities are also held for speculative purposes. Thus, stock levels respond strongly to prices, so the stock relationships were specified and estimated as

(10) qd(ic,it,irun,5) = bst(1,ic) + bst(2,ic)*rp(ic,it,irun) + bst(3,ic)*qd(ic,it-1,irun,5) + bst(4,ic)*time(it)

where qd(ic,it,irun,5) is ending stocks in year t.

Residual Use. For some crops (rice, peanuts, and cottonseed), supply and utilization data show a residual category, which is modeled as,

(11) qd(ic,it,irun,6) = brs(1,ic) + brs(2,ic)*rp(ic,it,irun) + brs(3,ic)*time(it)

where:

qd(ic,it,irun,6) = residual use.

Although quantities in this residual use category are never used, the level of the residual does respond negatively to the real price, and is thus viewed as another utilization (demand) category.

Market Clearing Identities

In supply and demand specification outlined above, supply generally depends on past prices, while demand depends on current prices. In simulating these econometrically estimated equations into the future, simulated prices are solved by simultaneously solving the market clearing identities

(12) qs(ic,it,irun) +qd(ic,it-1,irun,5) = qd(ic,it,irun,1) + qd(ic,it,irun,2) + qd(ic,it,irun,3) + qd(ic,it,irun,4) + qd(ic,it,irun,5) + qd(ic,it,irun,6)

where:

qs(ic,it,irun) = the quantity produced of crop ic in year it in simulation “irun”.

Production is defined to be qs(ic,it,irun) = acresh(ic,it,irun)*ey(ic,it,irun). The left hand side of the equal sign in (12) gives total supply (production plus beginning stocks), while the right-hand side of (12) gives total utilization, including ending stocks.

In the simulation model this set of simultaneous equations are numerically solved to get the market clearing prices in a given year. This process is continued, considering the dynamics of the model, indefinitely into the future.

Historical Observation Period

Many econometric relationships in the model were estimated with data for the 1975-1995 time period. However, where structural change was apparent, such as with stock holding behavior and international trade, some of the early years were dropped from statistical analysis so that the simulation model would better reflect the future.

Alternative Specifications Considered

Many different specifications of how farm programs influence crop acreage have been considered in the evolution of AGSIM©, including: (a) acreage depends on support price, (b) acreage depends on the maximum of expected market price and support price, (c) acreage depends on a weighted average of support and expected market prices, with weights based on program and non-program acreage of the crop, and (d) acreage depends on expected market price. Models for expected market price have considered complex distributed lags that go back several years in time, to a simple model that expected market price is actual price the previous year. Acreage equations have also been specified to depend on expected returns of: (1) all competing individual crops with no parameter restrictions, (2) all competing individual crops with full symmetry of cross-effects imposed on estimation, (3) major competing individual crops, and (4) a weighted average of all expected returns for all other crops. Many different ways of incorporating participation rates and acreage diverted into the model have also been considered. Several alternative functional forms (linear, log-linear, nonlinear share equations, asymptotic) have also been considered. Theoretical specifications considered have ranged from *ad hoc* models to very tightly specified and detailed theoretical economic models based on complex assumptions. The present model draws from economic theory (e.g. symmetry of cross-price effects in demand and homogeneity of degree zero of all supply and demand equations with respect to prices), but does not specify the model so tightly with untested assumptions and functional forms that empirical data has almost no role in the resulting estimates. Alternative estimation techniques, ranging from simultaneous equations techniques, to Zellner's seemingly unrelated regressions, to ordinary least squares regression have been used. The current version of AGSIM© reflects a degree of subjective judgement of what best reflects supply and demand of agricultural commodities based on microeconomic theory, traditional statistical criteria, and substantive direct contact with farmers and ranchers in most regions of the United States.

Baseline

The current version of AGSIM© is designed to estimate *changes* in the agricultural sector resulting from pesticide or other policy. Changes in economic variables are computed by comparing a policy simulation of the model with a baseline simulation of the model. For *ex post* (retrospective) evaluations, the baseline reflects actual farm programs, prices, acreages, etc. However, for *ex ante* evaluations,

AGSIM© is calibrated to an external baseline. The calibration is done by comparing an internally generated baseline to the external baseline and computing adjusted intercepts for all of the relevant demand and supply relationships in AGSIM©**.**

For the 1999 version of AGSIM© the externally specified year 2010 baseline is forecasted from the 2007 baseline reported by USDA (1988b). A few endogenous variables in AGSIM© were not included in the USDA baseline. In those cases, the 1997 FAPRI baseline was used (FAPRI, 1997).

It should be noted that the baseline is not especially critical to estimates of *changes* in the agricultural sector, except for the case of price support policy, which is not relevant here. That is, sensitivity analyses with previous versions of AGSIM© have shown that estimates of *changes* in variables are not very sensitive to baseline absolute values of variables. Use of the USDA baseline to the extent possible assures consistency with other governmental mandated agricultural policy analyses.

Regional Effects Sub-Model

AGSIM© subroutines are also available to combine AGSIM© output with production cost information to estimate net farm income impacts for the policy scenario at the regional level (or farm, representative farm, area or state level). Required information for this type of evaluation includes for each farm or area: (a) yield and cost changes (which often differ from the national yield and cost changes for the policy scenario), (b) baseline production costs, and(c) acreages of each crop. This information is combined with price impacts estimated with AGSIM©, and regional supply elasticities from a prior version of AGSIM© (or from other sources) to estimate net farm income changes for the farms or areas considered.

The conceptual foundation for regional evaluation in this version of AGSIM© begins with a net farm income formula,

$$\Pi_{ir} = \sum_{ic} A_{ic,ir} R_{ic,ir} \tag{13}$$

where:

Π_{ir} = net farm income in region ir,
$A_{ic,ir}$ = acreage harvested of the icth crop in that region, and
$R_{ic,ir}$ = per-acre net return in that region.

Based on equation (13), it can be shown that the theoretically appropriate formula for computing net farm income *changes* for different regional situations is:

$$\frac{\Delta \Pi_{ir}}{\Delta Z} \cong \sum_{ic} R_{ic,ir} \sum_{jc} \frac{\Delta A_{ic,ir}}{\Delta R_{jc,ir}} \frac{\Delta R_{jc,ir}}{\Delta Z} + \sum_{ic} A_{ic,ir} \sum_{jc} \frac{\Delta R_{ic,ir}}{\Delta Z} \tag{14}$$

where:

Δ represents a discrete change,
ΔZ represents the discrete policy change,
ic and jc are crop indices,

and other variables are as previously defined.

Equation (14) can be expressed in acreage elasticity (with respect to per-acre income) form,

$$(15) \qquad \frac{\Delta \Pi_{ir}}{\Delta Z} \cong \sum_{ic} R_{ic,ir} \sum_{jc} e_{ic,ij,ir} \frac{R_{ic,ir}}{A_{jc,ir}} \frac{\Delta R_{jc,ir}}{\Delta Z} + \sum_{ic} A_{ic,ir} \sum_{jc} \frac{\Delta R_{ic,ir}}{\Delta Z}$$

where:

$\epsilon_{ic,ij,ir}$ = elasticity of acreage of the icth crop in the irth region with respect to per-acre income of the jcth crop in that region.

The term $\Delta R_{ic,ij}/\Delta Z$ in equations (14) and (15) can be further expanded to give

$$(16) \qquad \frac{\Delta R_{ic,ir}}{\Delta Z} \cong P_{ic} \Delta Y_{ic,ir} + Y_{ic,ir} \Delta P_{ic} - \Delta C_{ic,ir}$$

Formula (15) along with (16) can be empirically implemented to estimate the change in regional (or farm, representative farm, area or state level) farm income with the following information for each region: (a) crop budgets, (b) the change in yield and cost associated with the policy in question, price impacts estimated with AGSIM©, and externally specified (from an older version of AGSIM©, from subjective estimates, or from the literature) elasticities.

The first term on the right-hand side of (14) and (15) represents the change in net income resulting from increased or decreased acreage, while the last term on the right-hand side of (14) and (15) represents the change in net farm income on existing acreage of crops in the region. Since acreage response is generally inelastic, the last term on the right-hand side of (14) and (15) dominates the change in net farm income in a region; thus, elasticities generally will not have a major impact on regional net farm income changes estimated with the above approach.

AGSIM© Output

The major outputs from AGSIM© are changes in crop acreage, production, price, income, foreign consumer benefits, domestic consumer benefits, and farm program costs. The traditional method of economic welfare analysis (which is based on the concept of economic surplus) of policy changes is used to compute the sum of changes in producer surplus (net farm income) plus changes to all consumers (changes in consumers surplus) plus any changes in farm program payments (zero under 1996 FAIR). To avoid the possibility of inappropriately comparing a baseline with a policy scenario that was actually based on another baseline, a single run of AGSIM© produces both the baseline tables and the policy scenario tables, then computes economic surplus and price changes based on these two runs of the model.

Output from each run of the model includes two sets of tables for each crop; one set of tables for supply variables and another set of tables for supply and utilization variables. Each table includes historical statistics as well as simulations into the future. These tables are constructed for the baseline and the policy scenario.

5.3 CONSUMER CLEANING COST SAVINGS

Particulate matter air pollution has been shown to result in dirtier clothes, which in turn results in higher annual cleaning costs for consumers. One benefit of reduced particulate matter, then, is the consequent reduction in cleaning costs for consumers. Several studies have provided estimates of the cost to households of PM soiling. The study that is cited by ESEERCO (1994) as one of the most sophisticated and is relied upon by EPA in its 1988 Regulatory Impact Analysis for SO_2 is Manuel et al. (1982). Using a household production function approach and household expenditure data from the 1972-73 Bureau of Labor Statistics Consumer Expenditure Survey for over twenty cities in the United States, Manuel et al. estimated the annual cost of cleaning per $\mu g/m^3$ PM per household as $1.55 ($0.59 per person times 2.63 persons per household). This estimate is low compared with others (e.g., estimates provided by Cummings et al. (1985) and Watson and Jaksch (1982) are about eight times and five times greater, respectively). The ESEERCO report notes, however, that the Manuel estimate is probably downward biased because it does not include the time cost of do-it-yourselfers. Estimating that these costs may comprise at least half the cost of PM-related cleaning costs, they double the Manuel estimate to obtain a point estimate of $3.10 (reported by ESEERCO in 1992 dollars as $2.70).

The Manuel et al. (1982) study measured particulate matter as TSP rather than PM_{10} or $PM_{2.5}$. If a one $\mu g/m^3$ increase in TSP causes $1.55 worth of cleaning expenses per household, the same unit dollar value can be used for PM_{10} (or $PM_{2.5}$) only if particle size doesn't matter -- i.e., only if particles of all sizes are equally soiling. Suppose, for example, that PM_{10} is 75% of TSP and that all particles are equally soiling. Then 75% of the damage caused by a one $\mu g/m^3$ increase in TSP is due to PM_{10}. This is (0.75)($1.55) = $1.16. However, this corresponds to a 0.75 $\mu g/m^3$ increase in PM_{10}. A one $\mu g/m^3$ increase in PM_{10} would therefore yield a dollar soiling damage of $1.16/0.75 = $1.55.

Suppose, however, that only PM_{10} matters. Then the $1.55 underestimates the impact of a one $\mu g/m^3$ increase in PM_{10}, because it corresponds to a less than one $\mu g/m^3$ increase in PM_{10} (e.g., a 0.75 $\mu g/m^3$ increase in PM_{10}). In this case, the correct unit value per unit of PM_{10} would be ($1.55)/0.75 = $2.07. If only PM_{10} matters, then either (1) the dollar value can be adjusted by dividing it by the percentage of TSP that is PM_{10} and PM_{10} can be used in the soiling damage function, or (2) the dollar value can be left unadjusted and TSP, rather than PM_{10}, can be used in the soiling damage function.

Finally, it is possible that, while both PM_{10} and $PM_{2.5}$ are components of TSP that cause consumer cleaning costs, the remaining portion of TSP has a greater soiling capability than either the PM_{10} or $PM_{2.5}$ component. In this case, using either PM_{10} or $PM_{2.5}$ air quality data with a household soiling function based on TSP would yield overestimates of the PM_{10}- or $PM_{2.5}$-related consumer cleaning costs avoided by reductions in concentration of these pollutants.

There is, however, insufficient information on the relative soiling capabilities of the different components of TSP. This analysis assumes that all components of TSP have an equivalent soiling capacity.

5.4 NITROGEN DEPOSITION

Excess nutrient loads, especially that of nitrogen, are responsible for a variety of adverse consequences to the health of estuarine and coastal waters, especially in the eastern United States. These effects include toxic and/or noxious algal blooms such as brown and red tides, low (hypoxic) or zero (anoxic) concentrations of dissolved oxygen in bottom waters, the loss of submerged aquatic vegetation due to the light-filtering effect of thick algal mats, and fundamental shifts in phytoplankton community structure. In order to model the impacts of nitrogen deposition on eastern estuaries, 10 eastern case study estuaries and two Gulf Coast estuaries have been chosen because of the availability of necessary data and their potential representativeness. Estimating nitrogen deposition in these 12 estuaries involves: (1) assigning county-level NO_x emissions to watershed-specific airsheds; and (2) calculating the change in nitrogen deposition to each estuary using both local area and broader regional deposition estimates to the watershed (kg of nitrogen deposited/ton of NO_x emitted). The nitrogen deposition rate estimates were derived by Pechan-Avanti (1999) using a methodology developed for the 1997 PM/Ozone/Regional Haze NAAQS RIA (U.S. EPA, 1997c).[50]

The benefits to surrounding communities of reduced nitrogen loadings are not included in the primary analysis. Instead, benefits attributed to the 12 case study estuaries are included as an alternative estimate of welfare benefits. The extrapolation of these benefits to 43 Eastern nutrient-sensitive estuaries is presented as a sensitivity analysis. Benefits due to reduced nitrogen deposition in the West are expected to be minimal, and are not calculated in this analysis.

Direct C-R functions relating deposited nitrogen and reductions in estuarine benefits are not available. The preferred WTP based measure of benefits depends on the availability of these C-R functions and on estimates of the value of environmental responses. Because neither appropriate C-R functions nor sufficient information to estimate the marginal value of changes in water quality exist at present, an avoided cost approach is used instead of WTP to generate estuary related benefits. This analysis uses the following data for each estuary: (1) total nitrogen load from all sources; (2) direct nitrogen load from atmospheric deposition to the estuary surface; (3) indirect nitrogen load from atmospheric deposition to the estuary watershed and subsequent pass-through to the estuary itself; (4) established nitrogen thresholds and reduction goals adopted by the community; and (5) costs associated with using agreed upon non-point water pollution control technologies.

Atmospheric nitrogen reductions are valued in this analysis on the basis of avoided costs associated with agreed upon controls of nonpoint water pollution sources. Benefits are estimated using an average, locally-based cost for nitrogen removal from water pollution (U.S. EPA, 1998). Valuation reflects water pollution control cost avoidance based on average cost/pound of current non-point source water pollution controls for nitrogen in three case study estuaries: Albemarle/Pamlico Sounds, Chesapeake Bay, and Tampa Bay. Taking the weighted cost/pound of these available controls assumes States will combine low cost and high cost controls.

In a recent advisory statement, the EPA's Science Advisory Board (SAB) was charged with reviewing the benefits methodology for the §812b report on the benefits and costs of the Clean Air Act Amendments. The SAB raised concerns about the use of the avoided cost approach to value reduced ecosystem damages. Specifically, they identified a key requirement which should be met in order for

[50]Further details on the estimation of nitrogen deposition are provided by EPA (1997b). Revisions to this methodology are described in U.S. EPA (1999)

avoided costs to approximate environmental benefits. This requirement is that there is a direct link between implementation of the air pollution regulation and the abandonment of a separate costly regulatory program by some other agency, i.e. a state environmental agency. Reductions in nitrogen deposition are expected to impact estuaries all along the eastern seaboard and the Gulf Coast. Many of the estuaries in these areas are currently being targeted by nitrogen reduction programs due to current impairment of estuarine water quality by excess nutrients. Some of the largest of these estuaries, including the Chesapeake Bay, have established goals for nitrogen reduction and target dates by which these goals should be achieved. Using the best and most easily implemented existing technologies, many of the estuaries will not be able to achieve the stated goals by the target dates. Meeting these additional reductions will require development of new technologies, implementation of costly existing technologies (such as stormwater controls), or use of technologies with significant implementation difficulties, such as agricultural best management practices (BMPs). Reductions in nitrogen deposition from the atmosphere will directly reduce the need for these additional costly controls. Thus while the final Tier II rule does not eliminate the need for nutrient management programs already in place, it may substitute for some of the incremental costs and programs (such as an agricultural BMP program) necessary to meet the nutrient reduction goals for each estuary.

It should be noted that avoided cost is only a proxy for benefits, and should be viewed as inferior to WTP based measures. Current research is underway to develop other approaches for valuing estuarine benefits, including contingent valuation and hedonic property studies. However, this research is still sparse, and does not contain sufficient information on the marginal WTP for changes in concentrations of nitrogen (or changes in water quality or water resources as a result of changes in nitrogen concentrations).

The fixed capital costs for non-point controls in the case study estuaries ranges from $0.75 to $55.59 per pound for agricultural and other rural best management practices and from $42.98 to $175.16 per pound for urban nonpoint source controls (stormwater controls, reservoir management, onsite disposal system changes, onsite BMPs). Using these as a base, the total fixed capital cost per pound (weighted by the ratio of the controlled nitrogen load to the estuary goal) is calculated for each of the case-study estuaries and applied in the valuation of their avoided nitrogen load controlled. The weighted capital costs per pound for the case-study estuaries are $40.95 for Albemarle-Pamlico Sounds, $26.79 for Chesapeake Bay, and $108.36 for Tampa Bay[51]. For the purposes of this analysis, EPA assumes that estuaries that have not yet established nutrient reduction goals will utilize the same types of nutrient management programs as projected for the case study estuaries. For the other nine estuaries, an average capital cost per pound of nitrogen (from the three case-estuaries) of $58.70/lb is calculated and applied; this cost may understate or overstate the costs associated with reductions in these other estuaries. The other nine estuaries generally represent smaller, more urban estuaries (like Tampa Bay), which typically have fewer technical and financial options available to control nitrogen loadings from nonpoint sources. This may result in higher control costs more similar to the Tampa Bay case. On the other hand, these estuaries may have opportunities to achieve additional point source controls at a lower cost. Also, increased public awareness of nutrification issues and technological innovation may, in the future, result in States finding lower cost solutions to nitrogen removal.

The benefits analysis assumed that the ten included East Coast estuaries are highly or moderately nutrient sensitive, and they represent approximately 45.46 percent of all estuarine watershed areas along

[51] The value for Tampa Bay is not a true weighted cost per pound, but a midpoint of a range of $71.89 to $144.47 developed by Apogee Research for the control possibilities (mostly urban BMPs) in the Tampa Bay estuary.

the East Coast.[52] Because data provided by the National Oceanic and Atmospheric Administration (NOAA) indicate that approximately 92.6 percent of the watershed and surface area of East Coast estuaries are highly or moderately nutrient sensitive, it is reasonable to expect that East Coast estuaries not included in this analysis would also benefit from reduced deposition of atmospheric nitrogen. Therefore, we scaled-up total benefits from the ten representative East Coast estuaries to include the remainder of the nutrient sensitive estuaries along the East Coast on the basis of estuary watershed plus water surface area. Since the ten estuaries are assumed to be nutrient sensitive and account for 48 percent of total eastern estuarine area, we scaled-up estimates by multiplying the estimate for the ten East Coast estuaries by 2.037 (equal to 92.6 percent divided by 45.46 percent). We then added this figure to the benefits estimated for the two Gulf Coast estuaries for a total benefits estimate for nitrogen deposition.

The analysis then annualized all capital cost estimates based on a seven percent interest rate and a typical implementation horizon for control strategies. Based on information from the three case study estuaries, this typically ranges from five to ten years. EPA has used the midpoint of 7.5 years for annualization, which yields an annualization factor of 0.1759. Non-capital installation costs and annual operating and maintenance costs are not included in these annual cost estimates. Depending upon the control strategy, these costs can be significant. Reports on the Albemarle-Pamlico Sounds indicate, for instance, that planning costs associated with control measures comprises approximately 15 percent of capital costs. Information received from the Association of National Estuary Programs indicates that operating and maintenance costs are about 30 percent of capital costs, and that permitting, monitoring, and inspections costs are about one to two percent of capital costs. For these reasons, the annual cost estimates may be understated.

[52] There are 43 East Coast estuaries of which ten were in the sample, and 31 Gulf of Mexico estuaries of which two are in the sample.

6 RESULTS

This chapter provides estimates of the magnitude and value of changes in selected health and welfare endpoints associated with Tier II-related changes in ambient Ozone and PM concentrations. The total dollar benefits associated with a given endpoint depend on how much the endpoint will change (e.g., how many premature deaths will be avoided) and how much each unit of change is worth (e.g., how much a premature death avoided is worth).

To place estimated incidence changes into context with predicted baseline incidence, Exhibit 6-1 displays the baseline incidence figures for those endpoints for which one can be calculated. Due to the nature of the endpoints, baseline incidence can only be calculated for Ozone- and PM-related health effects. In addition to baseline incidence, for each health effect, both the mean estimated incidence change and corresponding percent change between post-control incidence reductions and the predicted incidence baseline is presented.

Exhibits 6-2 and 6-3 present the primary incidence and benefit estimates associated with the primary scenario. A 5^{th} percentile, mean, and 95^{th} percentile estimate for both incidence and benefits is presented for each endpoint, as well as the simple mean benefit (calculated by multiplying the mean estimate of incidence by the corresponding mean valuation). Total benefits are also displayed, calculated by simply summing the simple mean of each endpoint.

Exhibit 6-4 displays alternative incidence and benefit calculations to those included in the primary analysis. Where possible, a 5^{th} percentile, mean, and 95^{th} percentile estimate for incidence and/or benefits is presented for each alternative endpoint. Exhibit 6-5 presents the aggregate uncertainty results (5^{th}, mean, and 95^{th} percentiles) for PM- and ozone-related benefits, as well as for total benefits (PM + ozone).

Exhibit 6-1 Baseline Percentages

Endpoint	Reference	2030 Control Scenario	
		Mean	% of Baseline
PM-RELATED BASELINE PERCENTAGES			
Ages 30+	Pope et al. (1995)	4,307	0.161%
Chronic Bronchitis	Pooled Analysis	2,296	0.308%
Respiratory-Related	Pooled Analysis	1,162	0.028%
Cardiovascular-Related	Pooled Analysis	485	0.008%
Asthma-Related ER Visits	Schwartz et al. (1993)	899	0.088%
Acute Bronchitis	Dockery et al. (1996)	7,933	0.732%
Upper Respiratory Symptoms	Pope et al. (1991)	86,476	0.067%
Lower Respiratory Symptoms	Schwartz et al. (1994)	87,123	0.512%
Shortness of Breath	Ostro et al. (1995)	17,434	0.280%
Work Loss Days	Ostro (1987)	682,898	0.133%
MRAD/Any-of-19	Pooled Analysis	3,628,527	0.214%
OZONE-RELATED BASELINE PERCENTAGES			
Chronic Asthma	McDonnell et al. (1999)	432	0.196%
Respiratory-Related Hospital Admissions	Pooled Analysis	1,012	0.024%
Dysrhythmias Hospital Admissions	Burnett et al. (1999)	269	0.033%
Asthma-Related ER Visits	Pooled Analysis	346	0.034%
MRAD/Any-of-19	Pooled Analysis	2,226,463	0.132%

Exhibit 6-2 Estimated PM-Related Health and Welfare Benefits Associated with Air Quality Changes Resulting from the Final Tier II Rule 2030 Control Scenario

Endpoint	Reference	Avoided Incidence (cases/year)			Monetary Benefits (millions 1997$)			Simple Mean
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile	
MORTALITY								
Ages 30+	Pope et al. (1995)	2,671	4,307	5,891	$3,193	$23,370	$57,022	$23,375
CHRONIC ILLNESS								
Chronic Bronchitis	Pooled Analysis	610	2,296	4,066	$58	$727	$2,463	$728
HOSPITALIZATION								
Respiratory-Related	Pooled Analysis	364	1,162	2,052	$4	$11	$18	$11
Cardiovascular-Related	Pooled Analysis	141	485	1,062	$2	$7	$15	$7
Asthma-Related ER Visits	Schwartz et al. (1993)	406	899	1,424	$0.1	$0.3	$0.4	$0.3
MINOR ILLNESS								
Acute Bronchitis	Dockery et al. (1996)	-40	7,933	16,313	< $0.1	$0.4	$1.1	$0.4
Upper Respiratory Symptoms	Pope et al. (1991)	25,475	86,476	144,578	$0.5	$2	$4	$2
Lower Respiratory Symptoms	Schwartz et al. (1994)	39,947	87,123	131,148	$0.4	$1	$2	$1
Shortness of Breath	Ostro et al. (1995)	4,697	17,434	29,508	< $0.1	$0.1	$0.3	$0.1
Work Loss Days	Ostro (1987)	597,804	682,898	771,811	$61	$70	$79	$70
MRAD/Any-of-19	Pooled Analysis	3,034,085	3,628,527	4,177,213	$97	$173	$253	$173
WELFARE EFFECTS								
Recreational Visibility	Based on Chestnut and Rowe (1990)		Direct Economic Valuation		-	$371	-	$371
TOTAL PRIMARY PM-RELATED BENEFITS					$4,510	-	$58,675	**$24,739**

Exhibit 6-3 Estimated Ozone-Related Health and Welfare Benefits Associated with Air Quality Changes Resulting from the Final Tier II Rule 2030 Control Scenario

Endpoint	Reference	Avoided Incidence (cases/year)			Monetary Benefits (millions 1997$)			Simple Mean
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile	
CHRONIC ILLNESS								
Chronic Asthma	McDonnell et al. (1999)	98	432	757	$3	$13	$23	$13
HOSPITALIZATION								
Respiratory-Related	Pooled Analysis	165	1,012	1,826	$2	$11	$21	$11
Cardiovascular-Related								
Dyrhythmias	Burnett et al. (1999)	-11	269	538	$0	$2	$4	$2
Asthma-Related ER Visits	Pooled Analysis	109	346	551	$0.0	$0.1	$0.2	$0.1
MINOR ILLNESS								
MRAD/Any-of-19	Pooled Analysis	1,014,435	2,226,463	3,414,837	$39	$101	$182	$101
WELFARE EFFECTS								
Decreased Worker Productivity	Crocker & Horst (1981) and EPA (1994)	Direct Economic Valuation			-	$142	-	$142
Agriculture		Direct Economic Valuation			-	$217	-	$217
TOTAL PRIMARY OZONE-RELATED BENEFITS					$278	-	$688	**$486**

Exhibit 6-4 Alternative Benefit Calculations for the Tier II 2030 Control Scenario

Endpoint	Reference/Alternative Valuation	Avoided Incidence (cases/year)			Monetary Benefits (millions 1997$)			Simple Mean
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile	
PM-RELATED ALTERNATIVE CALCULATIONS								
Mortality								
Ages 30+	Dockery et al. (1993)	4,472	9,820	15,479	$6,864	$53,577	$134,024	$53,297
Life Years Lost, Ages:	Pope et al. (1995)				-	$11,949	-	$11,949
30-34		1,108	1,822	2,496	-	-	-	-
35-44		3,603	5,925	8,116	-	-	-	-
45-54		4,215	6,933	9,496	-	-	-	-
55-64		6,516	10,717	14,679	-	-	-	-
65-74		8,576	14,105	19,319	-	-	-	-
75-84		6,803	11,188	15,324	-	-	-	-
85+		3,789	6,232	8,536	-	-	-	-
Chronic Bronchitis	Reversals	532	2,002	3,527	$16	$281	$973	$281
Chronic Bronchitis	Cost-of-Illness Valuation	610	2,296	4,066	-	$188	-	$188
Visibility								
Recreational	All U.S. Class I Areas	Direct Economic Valuation			-	$553	-	$553
	Eastern U.S.	Direct Economic Valuation			-	$423	-	$423
Residential	Continental U.S.	Direct Economic Valuation			-	$557	-	$557
Household Soiling Damage	ESEERCO (1994)	Direct Economic Valuation			$61	$111	$201	$111
Nitrogen Deposition	12 Estuaries	Direct Economic Valuation			-	$161	-	$161

Exhibit 6-5 Measures of Aggregate Uncertainty in the Benefits Analysis

Benefits Aggregation	Monetary Benefits (millions 1997$)			
	5th %ile	Mean	95th %ile	Simple Mean
Total Ozone-Related Benefits	$278	$485	$688	$486
Total PM-Related Benefits	$4,510	$24,973	$58,675	$24,739
Total Tier II Primary Analysis Benefits (Ozone + PM)	$4,971	$25,458	$59,133	$25,225

7 REFERENCES

Abbey, D.E., R.J. Burchette, S.F. Knutsen, W.F. McDonnell, M.D. Lebowitz and P.L. Enright. 1998. Long-term particulate and other air pollutants and lung function in nonsmokers. American Journal of Respiratory and Critical Care Medicine. 158(1): 289-298.

Abbey, D.E., B.L. Hwang, R.J. Burchette, T. Vancuren and P.K. Mills. 1995a. Estimated Long-Term Ambient Concentrations of Pm(10) and Development of Respiratory Symptoms in a Nonsmoking Population. Archives of Environmental Health. 50(2): 139-152.

Abbey, D.E., P.K. Mills, F.F. Petersen and W.L. Beeson. 1991. Long-Term Ambient Concentrations of Total Suspended Particulates and Oxidants As Related to Incidence of Chronic Disease in California 7th-Day Adventists. Environmental Health Perspectives. 94(AUG): 43-50.

Abbey, D.E., B.E. Ostro, F. Petersen and R.J. Burchette. 1995b. Chronic Respiratory Symptoms Associated with Estimated Long-Term Ambient Concentrations of Fine Particulates Less Than 2.5 Microns in Aerodynamic Diameter (PM2.5) and Other Air Pollutants. J Expo Anal Environ Epidemiol. 5(2): 137-159.

Abbey, D.E., F. Petersen, P.K. Mills and W.L. Beeson. 1993. Long-Term Ambient Concentrations of Total Suspended Particulates, Ozone, and Sulfur Dioxide and Respiratory Symptoms in a Nonsmoking Population. Archives of Environmental Health. 48(1): 33-46.

Abt Associates Inc. 1996a. An Analysis of the Monetized Benefits Associated with National Attainment of Alternative Particulate Matter Standards in the Year 2007. Prepared for U.S. EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC. July 5.

Abt Associates Inc. 1996b. A Particulate Matter Risk Assessment for Philadelphia and Los Angeles. Prepared for U.S. EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC. July 3.

Abt Associates Inc. 1999. Co-Control Benefits of Greenhouse Gas Control Policies. Prepared for U.S. EPA, Office of Policy, under contract no. 68-W4-0029. Washington, DC. February.

Ackermann-Liebrich, U., P. Leuenberger, J. Schwartz, C. Schindler, C. Monn, C. Bolognini, J.P. Bongard, O. Brandli, G. Domenighetti, S. Elsasser, L. Grize, W. Karrer, R. Keller, H. KellerWossidlo, N. Kunzli, B.W. Martin, T.C. Medici, A.P. Perruchoud, M.H. Schoni, J.M. Tschopp, B. Villiger, B. Wuthrich, J.P. Zellweger and E. Zemp. 1997. Lung function and long term exposure to air pollutants in Switzerland. Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) Team. Am J Respir Crit Care Med. 155(1): 122-129.

Adams, P.F. and V. Benson. 1992. Current Estimates from the National Health Interview Survey, 1991. National Center for Health Statistics. Hyattsville, MD. Vital Health Statistics, Series 10, No. 184. December.

Adams, P.F. and M.A. Marano. 1995. Current Estimates from the National Health Interview Survey, 1994. National Center for Health Statistics. Hyattsville, MD. Vital Health Statistics, Series 10, No. 193. December.

Alberini, A., M. Cropper, T.T. Fu, A. Krupnick, J.T. Liu, D. Shaw and W. Harrington. 1997. Valuing health effects of air pollution in developing countries: The case of Taiwan. Journal of Environmental Economics and Management. 34(2): 107-126.

Anderson, H.R., A. Ponce de Leon, J.M. Bland, J.S. Bower and D.P. Strachan. 1996. Air Pollution and Daily Mortality in London: 1987-92. British Medical Journal. 312: 65-669.

Blumenschein, K. and M. Johannesson. 1998. Relationship between quality of life instruments, health state utilities, and willingness to pay in patients with asthma. Ann Allergy Asthma Immunol. 80(2): 189-94.

Bobak, M. and D.A. Leon. 1992. Air pollution and infant mortality in the Czech Republic, 1986-88. Lancet. 340(8826): 1010-4.

Burnett, R.T. 1999. Email to Donald R. McCubbin, Abt Associates Inc.

Burnett, R.T., S. Cakmak, J.R. Brook and D. Krewski. 1997. The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. Environ Health Perspect. 105(6): 614-20.

Burnett, R.T., M. Smith-Doiron, D. Stieb, S. Cakmak and J.R. Brook. 1999. Effects of particulate and gaseous air pollution on cardiorespiratory hospitalizations. Archives Environmental Health. 54(2): 130-139.

Butler, R.J. 1983. Wage and Injury Rate Responses to Shifting Levels of Workers' Compensation. In Safety and the Work Force. Worrall, J.D., Ed. Cornell University, ILR Press: Ithaca, NY.

CARB (California Air Resources Board). 1982. California Ambient Air Quality Standard for Particulate Matter. Sacramento, CA. December.

Chestnut, L.G. 1995. Dollars and Cents: The Economic and Health Benefits of Potential Particulate Matter Reductions in the United States. Prepared for American Lung Association.

Chestnut, L.G. 1997. Draft Memorandum: Methodology for Estimating Values for Changes in Visibility at National Parks. April 15.

Chestnut, L.G. and R.D. Rowe. 1990. Preservation Values for Visibility Protection at the National Parks: Draft Final Report. Prepared for U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Economic Analysis Branch. Research Triangle, NC. February, 16.

Cody, R.P., C.P. Weisel, G. Birnbaum and P.J. Lioy. 1992. The effect of ozone associated with summertime photochemical smog on the frequency of asthma visits to hospital emergency departments. Environ Res. 58(2): 184-94.

Collins, J.G. 1997. Prevalence of Selected Chronic Conditions: United States 1990-1992. National Center for Health Statistics. Hyattsville, MD. Vital Health Statistics, Series 10, No. 194.

Council of Economic Advisers. 1997. The Annual Report of the Council of Economic Advisers. In Economic Report of the President. U.S. Government Printing Office: Washington, DC.

Council of Economic Advisers. 1998. The Annual Report of the Council of Economic Advisers. In Economic Report of the President. U.S. Government Printing Office: Washington, DC.

Cousineau, J., R. Lacroix and A. Girard. 1988. Occupational Hazard and Wage Compensating Differentials. University of Montreal Working Paper.

Cousineau, J., R. Lacroix and A. Girard. 1992. Occupational Hazard and Wage Compensating Differentials. The Review of Economics and Statistics. 74: 166-169.

Crocker, T.D. and R.L. Horst, Jr. 1981. Hours of Work, Labor Productivity, and Environmental Conditions: A Case Study. The Review of Economics and Statistics. 63: 361-368.

Cropper, M.L. and A.J. Krupnick. 1990. The Social Costs of Chronic Heart and Lung Disease. Resources for the Future. Washington, DC. Discussion Paper QE 89-16-REV.

Cummings, R., H. Burness and R. Norton. 1985. Methods Development for Environmental Control Benefits Assessment, Volume V. Measuring Household Soiling Damages from Suspended Air Particulates, A Methodological Inquiry. Prepared for U.S. Environmental Protection Agency. Washington, DC.

Decisioneering. 1996. Crystal Ball: Forecasting and Risk Analysis for Spreadsheet Users: User Manual. Version 4.0. www.decisioneering .com.

Delfino, R.J., M.R. Becklake and J.A. Hanley. 1994. The relationship of urgent hospital admissions for respiratory illnesses to photochemical air pollution levels in Montreal. Environ Res. 67(1): 1-19.

Detels, R., D.P. Tashkin, J.W. Sayre, S.N. Rokaw, F.J. Massey, A.H. Coulson and D.H. Wegman. 1991. The Ucla Population Studies of Cord .10. a Cohort Study of Changes in Respiratory Function Associated With Chronic Exposure to Sox, Nox, and Hydrocarbons. American Journal of Public Health. 81(3): 350-359.

Dickie, M. and S. Gerking. 1987. Reconciling Averting Behavior and Contingent Valuation Benefit Estimates of Reducing Symptoms of Ozone Exposure (draft), as cited in Neumann, J.E., M. Dickie, and R.E. Unsworth. 1994. Prepared by Industrial Economics. Prepared for Jim DeMocker, U.S. EPA, Office of Air and Radiation. March 31.

Dillingham, A. 1985. The Influence of Risk Variable Definition on Value of Life Estimates. Economic Inquiry. 24: 277-294.

Dockery, D.W., J. Cunningham, A.I. Damokosh, L.M. Neas, J.D. Spengler, P. Koutrakis, J.H. Ware, M. Raizenne and F.E. Speizer. 1996. Health Effects of Acid Aerosols On North American Children - Respiratory Symptoms. Environmental Health Perspectives. 104(5): 500-505.

Dockery, D.W., C.A. Pope, X.P. Xu, J.D. Spengler, J.H. Ware, M.E. Fay, B.G. Ferris and F.E. Speizer. 1993. An association between air pollution and mortality in six U.S. cities. N Engl J Med. 329(24): 1753-1759.

Dockery, D.W., F.E. Speizer, D.O. Stram, J.H. Ware, J.D. Spengler and B.G. Ferris, Jr. 1989. Effects of Inhalable Particles on Respiratory Health of Children. Am Rev Respir Dis. 139: 587-594.

Ehrlich, I. and H. Chuma. 1990. A Model of the Demand For Longevity and the Value of Life Extension. Journal of Political Economy. 98(4): 761-782.

Elixhauser, A., R.M. Andrews and S. Fox. 1993. Clinical Classifications for Health Policy Research: Discharge Statistics by Principal Diagnosis and Procedure. U.S. Department of Health Services, Center for General Health Services Intramural Research, Agency for Health Care Policy and Research.

Empire State Electric Energy Research Corporation (ESEERCO). 1994. New York State Environmental Externalities Cost Study. Report 2: Methodology. Prepared by RCG/Hagler, Bailly, Inc. November.

FAPRI. 1997. U.S. Agricultural Outlook. FAPRI Staff Report No. 1-97. Obtained via http://www.fapri.missouri.edu.

Garen, J. 1988. Compensating Wage Differentials and the Endogeneity of Job Riskiness. The Review of Economics and Statistics. 70(1): 9-16.

Gegax, D., S. Gerking and W. Shulze. 1985. Perceived Risk and the Marginal Value of Safety. Working paper prepared for the U. S. Environmental Protection Agency.

Gegax, D., S. Gerking and W. Shulze. 1991. Perceived Risk and the Marginal Value of Safety. The Review of Economics and Statistics. 73(4): 589-596.

Gerking, S., M. DeHaan and W. Schulze. 1988. The Marginal Value of Job Safety: A Contingent Valuation Study. Journal of Risk and Uncertainty. 1: 185-199.

Graves, E.J. and B.S. Gillum. 1997. Detailed Diagnoses and Procedures, National Hospital Discharge Survey, 1994. National Center for Health Statistics. Hyattsville, MD. Vital Health Statistics, Series 13, No. 127. March.

Herzog, H.W., Jr., and A.M. Schlottmann. 1987. Valuing Risk in the Workplace: Market Price, Willingness to Pay, and the Optimal Provision of Safety. University of Tennessee Working Paper.

Herzog, H.W., Jr., and A.M. Schlottmann. 1990. Valuing Risk in the Workplace: Market Price, Willingness to Pay, and the Optimal Provision of Safety. The Review of Economics and Statistics. 72(3): 463-470.

Hoek, G., J.D. Schwartz, B. Groot and P. Eilers. 1997. Effects of ambient particulate matter and ozone on daily mortality in Rotterdam, The Netherlands. Arch Environ Health. 52(6): 455-63.

Holland, M., D. Forster and M. Wenborn. 1999. Economic Valuation of Proposals Under the UNECE Multi-Effects and Multi-Pollutant Protocol. Prepared for: European Commission, DGXI. Brussels and Luxembourg. AEAT-4587. January.

Industrial Economics Incorporated (IEc). 1992. Review of Existing Value of Life Estimates: Valuation Document. Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. November 6.

Industrial Economics Incorporated (IEc). 1993. Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. September 30.

Industrial Economics Incorporated (IEc). 1994. Linkage Between Health Effects Estimation and Morbidity Valuation in the Section 812 Analysis -- Draft Valuation Document. Memorandum to Jim DeMocker, U.S. Environmental Protection Agency, Office of Air and Radiation, Office of Policy Analysis and Review. Prepared by J.E. Neumann, M.T. Dickie, and R.E. Unsworth. March 31.

Ito, K. 1998. Email to Ellen Post, Abt Associates Inc.

Ito, K. and G.D. Thurston. 1996. Daily PM10/mortality associations: an investigations of at-risk subpopulations. Journal of Exposure Analysis and Environmental Epidemiology. 6(1): 79-95.

Jones-Lee, M.W. 1989. The Economics of Safety and Physical Risk. Basil Blackwell: Oxford.

Jones-Lee, M.W., M. Hammerton and P.R. Philips. 1985. The Value of Safety: Result of a National Sample Survey. Economic Journal. 95(March): 49-72.

Kennedy. 1990. A Guide to Econometrics. 2nd ed. MIT Press: Cambridge, MA.

Kinney, P.L., K. Ito and G.D. Thurston. 1995. A Sensitivity Analysis of Mortality Pm-10 Associations in Los Angeles. Inhalation Toxicology. 7(1): 59-69.

Kniesner, T.J. and J.D. Leeth. 1991. Compensating Wage Differentials for Fatal Injury Risk in Australia, Japan, and the United States. Journal of Risk and Uncertainty. 4(1): 75-90.

Krupnick, A.J. 1988. An Analysis of Selected Health Benefits from Reductions in Photochemical Oxidants in the Northeastern United States: Final Report. Prepared for U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Washington, DC. EPA Contract No. 68-02-4323. September.

Krupnick, A.J. and M.L. Cropper. 1992. The Effect of Information On Health Risk Valuations. Journal of Risk and Uncertainty. 5(1): 29-48.

Krupnick, A.J., W. Harrington and B. Ostro. 1990. Ambient Ozone and Acute Health Effects - Evidence From Daily Data. Journal of Environmental Economics and Management. 18(1): 1-18.

Krupnick, A.J. and R.J. Kopp. 1988. The Health and Agricultural Benefits of Reductions in Ambient Ozone in the United States. Resources for the Future. Washington, DC. Discussion Paper QE88-10. August.

Lang, C., G. Yarwood, F. Lalonde and R. Bloxam. 1995. Environmental and Health Benefits of Cleaner Vehicles and Fuels. Prepared for: Canadian Council of Ministers of the Environment Task Force on Cleaner Vehicles and Fuels. Winnipeg, Manitoba. October.

Lee, E.H. and W.E. Hogsett. 1996. Methodology for Calculating Inputs for Ozone Secondary Standard Benefits Analysis: Part II. Prepared for U.S. EPA, Office of Air Quality Planning and Standards. March.

Leigh, J.P. 1987. Gender, Firm Size, Industry and Estimates of the Value-of-Life. Journal of Health Economics. 6: 255-273.

Leigh, J.P. and R.N. Folsom. 1984. Estimates of the Value of Accident Avoidance at the Job Depend on Concavity of the Equalizing Differences Curve. The Quarterly Review of Economics and Business. 24(1): 56-66.

Lipfert, F.W. 1993. A Critical Review of Studies of the Association Between Demands For Hospital Services and Air Pollution. Environmental Health Perspectives. 101(S2): 229-268.

Loehman, E.T., S.V. Berg, A.A. Arroyo, R.A. Hedinger, J.M. Schwartz, M.E. Shaw, R.W. Fahien, V.H. De, R.P. Fishe, D.E. Rio, W.F. Rossley and A.E.S. Green. 1979. Distributional Analysis of Regional Benefits and Cost of Air Quality Control. Journal of Environmental Economics and Management. 6: 222-243.

Loehman, E.T. and V.H. De. 1982. Application of Stochastic Choice Modeling to Policy Analysis of Public Goods. The Review of Economics and Statistics. 64(3): 474-480.

Loomis, D., M. Castillejos, D.R. Gold, W. McDonnell and V.H. Borja-Aburto. 1999. Air pollution and infant mortality in Mexico City. Epidemiology. 10(2): 118-23.

Loomis, D.P., V.H. Borja-Aburto, S.I. Bangdiwala and C.M. Shy. 1996. Ozone Exposure and Daily Mortality in Mexico City: A Time-Series Analysis. Health Effects Institute. Cambridge, MA. Research Report Number 75. October.

Malm, W.C., J. Sisler, D. Huffman, R. Eldred and T. Cahill. 1994. Spatial and Seasonal Trends in Particle Concentration and Optical Extinction in the United States. Journal of Geophysical Research. 99(D1): 1247-1370.

Manuel, E.H., R.L. Horst, K.M. Brennan, W.N. Lanen, M.C. Duff and J.K. Tapiero. 1982. Benefits Analysis of Alternative Secondary National Ambient Air Quality Standards for Sulfur Dioxide and Total Suspended Particulates, Volumes I-IV. Prepared for U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Research Triangle Park, NC.

Marin, A. and G. Psacharopoulos. 1982. The Reward for Risk in the Labor Market: Evidence from the United Kingdom and a Reconciliation with Other Studies. Journal of Political Economy. 90(4): 827-853.

Mathtech Inc. 1997. Draft: Volume II Technical Support Document for Ozone NAAQS Analysis: Benefit Methdology. Prepared for Science Applications International Corporation for the U.S. EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC.

McClelland, G., W. Schulze, D. Waldman, J. Irwin, D. Schenk, T. Stewart, L. Deck and M. Thayer. 1991. Valuing Eastern Visibility: A Field Test of the Contingent Valuation Method. Prepared for U.S. Environmental Protection Agency, Office of Policy, Planning and Evaluation. June.

McDonnell, W.F., D.E. Abbey, N. Nishino and M.D. Lebowitz. 1999. Long-term ambient ozone concentration and the incidence of asthma in nonsmoking adults: the AHSMOG study. Environ Res. 80(2 Pt 1): 110-21.

Medical Center Information Systems: Duke University Health Systems. 1999. ICD-9-CM. http://dumccss.mc.duke.edu/standards/termcode/icd9/index.html. November 5.

Miller, T. and J. Guria. 1991. The Value of Statistical Life in New Zealand. Report to the New Zealand Ministry of Transport, Land Transport Division.

Mitchell, R.C. and R.T. Carson. 1986. The Use of Contingent Valuation Data for Benefit/Cost Analysis in Water Pollution Control. Draft report submitted by Resources for the Future to Environmental Protection Agency, Office of Policy Analysis. Washington, DC. September.

Mood, A.M., F.A. Graybill and D.C. Boes. 1974. Introduction to the Theory of Statistics. 3rd ed. McGraw Hill Book Company: New York.

Moolgavkar, S.H., E.G. Luebeck and E.L. Anderson. 1997. Air pollution and hospital admissions for respiratory causes in Minneapolis St. Paul and Birmingham. Epidemiology. 8(4): 364-370.

Moolgavkar, S.H., E.G. Luebeck, T.A. Hall and E.L. Anderson. 1995. Air Pollution and Daily Mortality in Philadelphia. Epidemiology. 6(5): 476-484.

Moore, M.J. and W.K. Viscusi. 1988. Doubling the Estimated Value of Life: Results Using New Occupational Fatality Data. Journal of Policy Analysis and Management. 7(3): 476-490.

Morgan, G., S. Corbett, J. Wlodarczyk and P. Lewis. 1998. Air pollution and daily mortality in Sydney, Australia, 1989 through 1993. Am J Public Health. 88(5): 759-64.

National Center for Health Statistics. 1999. National Vital Statistics Reports. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics. Washington, DC. Volume 47, Number 19. June 30.

National Heart, L., and Blood Institute. 1997. Guidelines for the Diagnosis and Management of Asthma: Expert Panel Report 2. National Institutes of Health. Bethesda, MD. NIH Publication No. 97-4051. July.

Neas, L.M., D.W. Dockery, J.H. Ware, J.D. Spengler, B.G. Ferris and F.E. Speizer. 1994. Concentration of Indoor Particulate Matter As a Determinant of Respiratory Health in Children. American Journal of Epidemiology. 139(11): 1088-1099.

O'Connor, R.M. and G.C. Blomquist. 1997. Measurement of Consumer-Patient Preferences Using a Hybrid Contingent Valuation Method. Journal of Health Economics. 16: 667-683.

Olson, C.A. 1981. An Analysis of Wage Differentials Received by Workers on Dangerous Jobs. Journal of Human Resources. 16: 167-185.

Ostro, B.D. 1987. Air Pollution and Morbidity Revisited: A Specification Test. Journal of Environmental Economics and Management. 14: 87-98.

Ostro, B.D., M.J. Lipsett and N.P. Jewell. 1989a. Predicting Respiratory Morbidity From Pulmonary Function Tests - a Reanalysis of Ozone Chamber Studies. Japca. 39(10): 1313-1318.

Ostro, B.D., M.J. Lipsett, J.K. Mann, H. Braxtonowens and M.C. White. 1995. Air Pollution and Asthma Exacerbations Among African-American Children in Los Angeles. Inhalation Toxicology. 7(5): 711-722.

Ostro, B.D., M.J. Lipsett, M.B. Wiener and J.C. Selner. 1991. Asthmatic Responses to Airborne Acid Aerosols. Am J Public Health. 81(6): 694-702.

Ostro, B.D. and S. Rothschild. 1989b. Air Pollution and Acute Respiratory Morbidity - an Observational Study of Multiple Pollutants. Environ Res. 50(2): 238-247.

Ostro, B.D., J.M. Sanchez, C. Aranda and G.S. Eskeland. 1996. Air Pollution and Mortality - Results From a Study of Santiago, Chile. J Expo Anal Environ Epidemiol. 6(1): 97-114.

Pechan -Avanti Group. 1999. Emissions and Air Quality Impacts of Proposed Motor Vehicle Tier 2 and Fuel Sulfur Standards. Prepared for U.S. EPA, Office of Air Quality Planning and Standards, Innovative Strategies and Economics Group. Springfield, VA. Prepared under EPA Contract No. 68-D9-8052. January.

Pechan-Avanti Group. 1999. Emissions and Air Quality Impacts of Final Motor Vehicle Tier 2 and Fuel Sulfur Standards: Draft Report. Prepared for U.S. EPA, Office of Air Quality Planning and Standards, Innovative Strategies and Economics Group. Springfield, VA. Pechan Report No. 99.10.001/9004.223. October.

Pereira, L.A.A., D. Loomis, G.M.S. Conceicao, A.L.F. Braga, R.M. Arcas, H.S. Kishi, R.M. Singer, G.M. Bohm and P.H.N. Saldiva. 1998. Association between air pollution and intrauterine mortality in Sao Paulo, Brazil. Environmental Health Perspectives. 106(6): 325-329.

Pope, C.A., D.W. Dockery, J.D. Spengler and M.E. Raizenne. 1991. Respiratory Health and Pm10 Pollution - a Daily Time Series Analysis. American Review of Respiratory Disease. 144(3): 668-674.

Pope, C.A., J. Schwartz and M.R. Ransom. 1992. Daily Mortality and PM10 Pollution in Utah Valley. Archives of Environmental Health. 47(3): 211-217.

Pope, C.A., M.J. Thun, M.M. Namboodiri, D.W. Dockery, J.S. Evans, F.E. Speizer and C.W. Heath. 1995. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Respir Crit Care Med. 151(3): 669-674.

Portney, P.R. and J. Mullahy. 1990. Urban Air Quality and Chronic Respiratory Disease. Regional Science and Urban Economics. 20(3): 407-418.

Richards, W., S.P. Azen, J. Weiss, S. Stocking and J. Church. 1981. Los Angeles air pollution and asthma in children. Ann Allergy. 47(5 Pt 1): 348-54.

Rossi, G., M.A. Vigotti, A. Zanobetti, F. Repetto, V. Gianelle and J. Schwartz. 1999. Air pollution and cause-specific mortality in Milan, Italy, 1980-1989. Arch Environ Health. 54(3): 158-64.

Rowe, R.D. and L.G. Chestnust. 1986. Oxidants and Asthmatics in Los Angeles: A Benefits Analysis -- Executive Summary. Prepared for U.S. Environmental Protection Agency, Office of Policy Analysis. Prepared by Energy and Resource Consultants, Inc. Washington, DC. EPA-230-09-86-018. March.

Saldiva, P.H.N., A. Lichtenfels, P.S.O. Paiva, I.A. Barone, M.A. Martins, E. Massad, J.C.R. Pereira, V.P. Xavier, J.M. Singer and G.M. Bohm. 1994. Association Between Air Pollution and Mortality Due to Respiratory Diseases in Children in Sao Paulo, Brazil - a Preliminary Report. Environ Res. 65(2): 218-225.

Samet, J.M., S.L. Zeger, J.E. Kelsall, J. Xu and L.S. Kalkstein. 1997. Air Pollution, Weather, and Mortality in Philadelphia 1973-1988. Health Effects Institute. Cambridge, MA. March.

Schwartz, J. 1993. Particulate Air Pollution and Chronic Respiratory Disease. Environ Res. 62: 7-13.

Schwartz, J. 1994a. Air Pollution and Hospital Admissions For the Elderly in Birmingham, Alabama. American Journal of Epidemiology. 139(6): 589-598.

Schwartz, J. 1994b. Air Pollution and Hospital Admissions For the Elderly in Detroit, Michigan. American Journal of Respiratory and Critical Care Medicine. 150(3): 648-655.

Schwartz, J. 1994c. PM(10) Ozone, and Hospital Admissions For the Elderly in Minneapolis St Paul, Minnesota. Archives of Environmental Health. 49(5): 366-374.

Schwartz, J. 1994d. What Are People Dying of On High Air Pollution Days. Environmental Research. 64(1): 26-35.

Schwartz, J. 1995. Short term fluctuations in air pollution and hospital admissions of the elderly for respiratory disease. Thorax. 50(5): 531-538.

Schwartz, J. 1996. Air pollution and hospital admissions for respiratory disease. Epidemiology. 7(1): 20-28.

Schwartz, J. 1997. Air pollution and hospital admissions for cardiovascular disease in Tucson. Epidemiology. 8(4): 371-377.

Schwartz, J. 1999. Air pollution and hospital admissions for heart disease in eight U.S. counties. Epidemiology. 10(1): 17-22.

Schwartz, J. and D.W. Dockery. 1992. Particulate Air Pollution and Daily Mortality in Steubenville, Ohio. American Journal of Epidemiology. 135(1): 12-19.

Schwartz, J., D.W. Dockery and L.M. Neas. 1996. Is Daily Mortality Associated Specifically With Fine Particles. Journal of the Air & Waste Management Association. 46(10): 927-939.

Schwartz, J., D.W. Dockery, L.M. Neas, D. Wypij, J.H. Ware, J.D. Spengler, P. Koutrakis, F.E. Speizer and B.G. Ferris. 1994. Acute Effects of Summer Air Pollution On Respiratory Symptom Reporting in Children. Am J Respir Crit Care Med. 150(5): 1234-1242.

Schwartz, J. and R. Morris. 1995. Air Pollution and Hospital Admissions For Cardiovascular Disease in Detroit, Michigan. American Journal of Epidemiology. 142(1): 23-35.

Schwartz, J., D. Slater, T.V. Larson, W.E. Pierson and J.Q. Koenig. 1993. Particulate air pollution and hospital emergency room visits for asthma in Seattle. Am Rev Respir Dis. 147(4): 826-31.

Sheppard, L., D. Levy, G. Norris, T.V. Larson and J.Q. Koenig. 1999. Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington, 1987-1994. Epidemiology. 10(1): 23-30.

Smith, D.H., D.C. Malone, K.A. Lawson, L.J. Okamoto, C. Battista and W.B. Saunders. 1997. A national estimate of the economic costs of asthma. Am J Respir Crit Care Med. 156(3 Pt 1): 787-93.

Smith, R.S. 1974. The Feasibility of an 'Injury Tax' Approach to Occupational Safety. Law and Contemporary Problems. 38(4): 730-744.

Smith, R.S. 1976. The Occupational Safety and Health Act: Its Goals and Achievements. American Enterprise Institute. Washington, DC.

Smith, V.K. 1983. The Role of Site and Job Characteristics in Hedonic Wage Models. Journal of Urban Economics. 13: 296-321.

Smith, V.K. and C. Gilbert. 1984. The Implicit Risks to Life: A Comparative Analysis. Economics Letters. 16: 393-399.

Smith, V.K., G. Van Houten and S. Pattanayak. 1999. Benefits Transfer as Preference Calibration. Resources for the Future. Washington, DC. Working Paper 99-36. May.

Spix, C., J. Heinrich, D. Dockery, J. Schwartz, G. Volksch, K. Schwinkowski, C. Collen and H.E. Wichmann. 1993. Air Pollution and Daily Mortality in Erfurt, East-Germany, 1980-1989. Environmental Health Perspectives. 101(6): 518-526.

Stieb, D.M., R.T. Burnett, R.C. Beveridge and J.R. Brook. 1996. Association between ozone and asthma emergency department visits in Saint John, New Brunswick, Canada. Environmental Health Perspectives. 104(12): 1354-1360.

Taylor, C.R., K.H. Reichelderfer and S.R. Johnson. 1993. Agricultural Sector Models for the United States: Descriptions and Selected Policy Applications. Iowa State University Press: Ames, IA.

Thurston, G.D., K. Ito, C.G. Hayes, D.V. Bates and M. Lippmann. 1994. Respiratory hospital admissions and summertime haze air pollution in Toronto, Ontario: consideration of the role of acid aerosols. Environ Res. 65(2): 271-290.

Thurston, G.D., K. Ito, P.L. Kinney and M. Lippmann. 1992. A multi-year study of air pollution and respiratory hospital admissions in three New York State metropolitan areas: results for 1988 and 1989 summers. J Expo Anal Environ Epidemiol. 2(4): 429-450.

Tolley, G.S. and et al. 1986. Valuation of Reductions in Human Health Symptoms and Risks. Prepared for U.S. Environmental Protection Agency. January.

U.S. Bureau of Economic Analysis. 1995. BEA Regional Projections to 2045: Volume 1, States. U.S. Department of Commerce. Washington, DC. July.

U.S. Bureau of the Census. 1992. Statistical Abstract of the United States: 1992. 112 ed. Washington, DC.

U.S. Bureau of the Census. 1997. Statistical Abstract of the United States: 1997. 117 ed. Washington, DC.

U.S. Bureau of the Census. 1998. Statistical Abstract of the United States: 1998. 118 ed. Washington, DC.

U.S. Centers for Disease Control. 1999. CDC Wonder. http://wonder.cdc.gov/. May.

U.S. Department of Agriculture. 1984. Usual Planting and Harvesting Dates for U.S. Field Crops.

U.S. Department of Agriculture. 1988a. County Crops Dataset. Electronic files. Obtained via http://usda.mannlib.cornell.edu/data-sets/crops/9X100/F1,.

U.S. Department of Agriculture. 1988b. USDA Agricultural Baseline Projections to 2007. World Agricultural Outlook Board, Office of Chief Economist, U.S. Department of Agriculture. Prepared by InterInteragency Agricultural Projections Committee. Staff Report No. WAOB-98-1. Obtained via http://www.econ.ag.gov/briefing/baseline.

U.S. EPA. 1986. Review of the National Ambient Air Quality Standards for Particulate Matter: Updated Assessment of Scientific and Technical Information Addendum to the 1982 OAQPS Staff Paper. U.S. EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC. EPA 450/05-86-012.

U.S. EPA. 1993. PRZM-2. A model for predicting pesticide fate in the crop root and unsaturated soil zones. User Manual for Release 2.0. EPA/600/R-93/046.

U.S. EPA. 1994. Documentation for Oz-One Computer Model (Version 2.0). Prepared for U.S. EPA, Office of Air Quality Planning and Standards. Prepared by Mathtech, Inc., under Contract No. 68D30030, WA 1-29. Research Triangle Park, NC. August.

U.S. EPA. 1996a. Air Quality Criteria for Ozone and Related Photochemical Oxidants. Volume III. U.S. EPA, Office of Research and Development. Washington, DC. EPA-/600/P-93/004cF. July.

U.S. EPA. 1996b. Review of National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information. OAQPS Staff Paper. U.S. EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC. EPA-452\R-96-007. June.

U.S. EPA. 1997a. The Benefits and Costs of the Clean Air Act: 1970 to 1990. U.S. EPA, Office of Air and Radiation, Office of Policy, Planning and Evaluation. Washington, DC. EPA 410-R-97-002. October.

U.S. EPA. 1997b. Benefits of Reducing Deposition of Atmospheric Nitrogen in Estuarine and Coastal Waters. U.S. EPA, Office of Air Quality Planning and Standards. July.

U.S. EPA. 1997c. Regulatory Impact Analyses for the Particulate Matter and Ozone National Ambient Air Quality Standards and Proposed Regional Haze Rule. U.S. EPA, Office of Air Quality Planning and Standards. Research Triangle Park, NC. July.

U.S. EPA. 1998. The Regional NOx SIP Call & Reduced Atmospheric Deposition of Nitrogen: Benefits to Selected Estuaries. September 22.

U.S. EPA. 1999a. The Benefits and Costs of the Clean Air Act: 1990 to 2010: EPA Report to Congress. U.S. EPA, Office of Air and Radiation, Office of Policy. Washington, DC. EPA 410-R-99-001.

U.S. EPA. 1999b. An SAB Advisory: The Clean Air Act Section 812 Prospective Study Health and Ecological Initial Studies. Prepared by the Health and Ecological Effects SubCommittee (HEES) of the Advisory Council on the Clean Air Compliance Analysis, Science Advisory Board, U.S. Environmental Protection Agency. Washington, DC. EPA-SAB-Council-ADV-99-005. February.

Verhoeff, A.P., G. Hoek, J. Schwartz and J.H. Vanwijnen. 1996. Air Pollution and Daily Mortality in Amsterdam. Epidemiology. 7(3): 225-230.

Violette, D.M. and L.G. Chestnut. 1983. Valuing Reductions in Risks: A Review of the Empirical Estimates. Prepared for U.S. Environmental Protection Agency. Washington DC. EPA-230-05-83-002.

Viscusi, W.K. 1978. Labor Market Valuations of Life and Limb: Empirical Estimates and Policy Implications. Public Policy. 26(3): 359-386.

Viscusi, W.K. 1979. Employment Hazards: An Investigation of Market Performance. Harvard University Press: Cambridge.

Viscusi, W.K. 1981. Occupational Safety and Health Regulation: Its Impact and Policy Alternatives. In Research in Public Policy Analysis and Management. Crecine, J., Ed. JAI Press: Greenwich, CT. p. 281-299.

Viscusi, W.K. 1992. Fatal Tradeoffs: Public and Private Responsibilities for Risk. Oxford University Press: New York.

Viscusi, W.K., W.A. Magat and J. Huber. 1991. Pricing Environmental Health Risks - Survey Assessments of Risk - Risk and Risk - Dollar Trade-Offs For Chronic Bronchitis. Journal of Environmental Economics and Management. 21(1): 32-51.

Wang, X., H. Ding, L. Ryan and X. Xu. 1997. Association between air pollution and low birth weight: a community- based study. Environ Health Perspect. 105(5): 514-20.

Watson, W. and J. Jaksch. 1982. Air Pollution: Household Soiling and Consumer Welfare Losses. Journal of Environmental Economics and Management. 9: 248-262.

Weisel, C.P., R.P. Cody and P.J. Lioy. 1995. Relationship between summertime ambient ozone levels and emergency department visits for asthma in central New Jersey. Environ Health Perspect. 103 Suppl 2: 97-102.

Wessex, I. 1994. PRO/FILER, US Demographics and ZIPS: Summary Tape File 1A. [CD-ROM]. Winnetka, Illinois.

Whittemore, A.S. and E.L. Korn. 1980. Asthma and Air Pollution in the Los Angeles Area. Am J Public Health. 70: 687-696.

Woodruff, T.J., J. Grillo and K.C. Schoendorf. 1997. The relationship between selected causes of postneonatal infant mortality and particulate air pollution in the United States. Environmental Health Perspectives. 105(6): 608-612.

Zeger, S.L., F. Dominici and J. Samet. 1999. Harvesting-resistant estimates of air pollution effects on mortality. Epidemiology. 10(2): 171-5.

APPENDIX A: RESULTS FOR SUPPLEMENTARY CALCULATIONS AND SENSITIVITY ANALYSES

Exhibit A-1 Supplemental Benefit Estimates for the Final Tier II Rule 2030 Control Scenario

			Avoided Incidence (cases/year)			Monetary Benefits (millions 1997$)			Simple Mean
Endpoint	**Reference**	**Pollutant**	**5th %ile**	**Mean**	**95th %ile**	**5th %ile**	**Mean**	**95th %ile**	
Short-Term Mortality	Schwartz et al. (1996)	PM	983	1,158	1,322	$980	$6,318	$14,493	$6,283
Post-Neonatal Mortality	Woodruff et al. (1997)	PM	7	13	20	$9	$71	$176	$71
Cardiac	Burnett et al. (1997)	Ozone	5,266	9,699	13,873	$71	$130	$188	$130
Moderate/Worse Asthma	Ostro et al. (1991)	PM	15,895	79,422	142,473	$0	$3	$7	$3
Asthma Attacks	Whittemore and Korn (1980)	PM	29,560	76,866	127,718	$1	$3	$6	$3
Asthma Attacks	Whittemore and Korn (1980)	Ozone	64,505	188,069	311,118	$2	$8	$15	$7
Restricted Activity Days	Ostro (1987)	PM	1,726,662	1,923,255	2,120,714	-	-	-	

Exhibit A-2 Sensitivity Analysis Results for the Tier II 2030 Control Scenario

Endpoint	Reference/Alternative Valuation	Avoided Incidence (cases/year)			Monetary Benefits (millions 1997$)			Simple Mean
		5th %ile	Mean	95th %ile	5th %ile	Mean	95th %ile	
MORTALITY								
Mortality Lags:								
No Lag		-	4,307	-	-	$25,387	-	-
8 Year	Incidence Occurs 8th Year	-	4,307	-	-	$18,042	-	-
15 Year	Incidence Occurs 15th Year	-	4,307	-	-	$12,822	-	-
15 Year	Incidence Skewed Early	-	4,307	-	-	$22,656	-	-
15 Year	Incidence Skewed Late	-	4,307	-	-	$14,753	-	-
WELFARE EFFECTS								
Nitrogen Deposition	All Eastern Estuaries	Direct Economic Valuation			-	$307	-	$307

Exhibit A-3 Sensitivity Analysis: Effect of Thresholds on Estimated PM-Related Mortality Based on Pope et al. (1995)

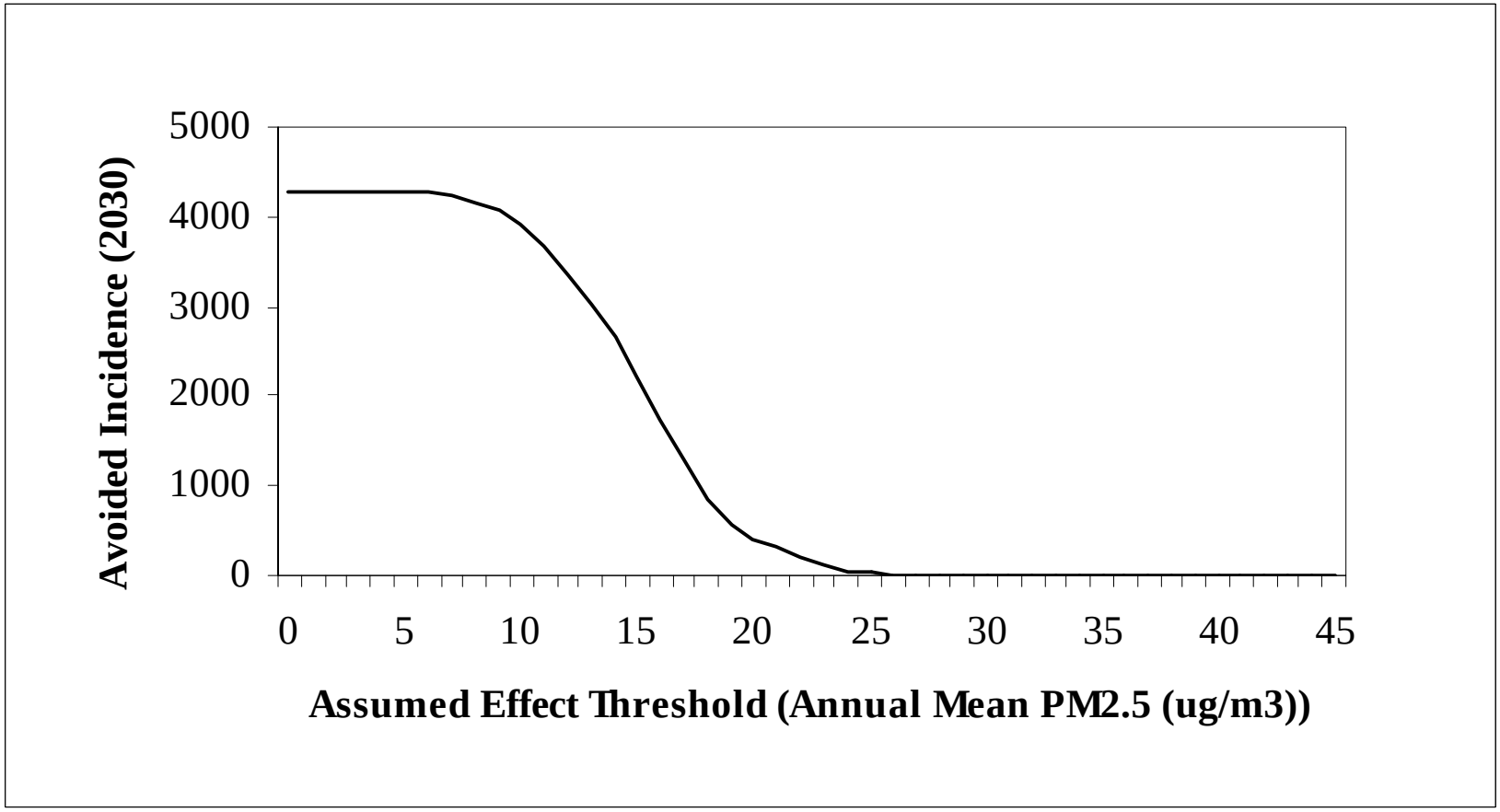

Exhibit A-4 Underlying Estimates and Weights for Pooled Estimate of PM-Related Respiratory Hospital Admissions

Study	Ages affected	Study weights	5th %ile	mean	95th %ile
Burnett et al. (1997), Toronto	all ages	0.03	-34	32	95
Burnett et al. (1999), Toronto	all ages	0.01	1,310	2,495	4,491
Thurston et al. (1994), Toronto	all ages	0.02	-457	667	1,743
Moolgavkar et al. (1997), Twin Cities	>64	0.19	307	751	1,277
Schwartz (1994c), Twin Cities	>64	0.12	1,037	1,624	2,271
Schwartz (1994a), Birmingham	>64	0.18	853	1,308	1,852
Schwartz (1994b), Detroit	>64	0.23	661	1,031	1,524
Schwartz (1996), Spokane	>64	0.14	827	1,364	1,947
Schwartz (1996), New Haven	>64	0.06	499	1,400	2,319
Schwartz (1996), Tacoma	>64	0.03	343	1,699	3,079
Pooled estimate of respiratory hospital admissions			362	1,162	2,052

Exhibit A-5 Underlying Estimates and Weights for Pooled Estimate of Ozone-Related Respiratory Hospital Admissions

Study	Ages affected	Study weights	5th %ile	mean	95th %ile
Burnett et al. (1997), Toronto	all ages	0.01	4,040	6,148	8,258
Burnett et al. (1999), Toronto	all ages	0.01	1,065	1,508	1,955
Thurston et al. (1994), Toronto	all ages	0.01	89	1,170	2,317
Moolgavkar et al. (1997), Twin Cities	>64	0.33	509	948	1,382
Schwartz (1994c), Twin Cities	>64	0.28	-20	467	965
Schwartz (1994b), Detroit	>64	0.26	909	1,399	1,893
Schwartz (1996), New Haven	>64	0.08	155	994	1,847
Schwartz (1996), Tacoma	>64	0.02	1,147	2,691	4,280
Pooled estimate of respiratory hospital admissions			165	1,012	1,826

Exhibit A-6 Underlying Estimates and Weights for Pooled Estimate of PM-Related Cardiovascular Hospital Admissions

Study	Ages Affected	Study Weights	5th %ile	Mean	95th %ile
Burnett et al. (1997), Toronto	all ages	0.33	117	229	347
Burnett et al. (1999), Toronto	all ages	0.03	-44	346	755
Schwartz and Morris (1995), Detroit	>64	0.30	213	421	628
Schwartz (1999), 8 US Counties	>64	0.25	472	751	1,041
Schwartz (1997), Tucson	>64	0.09	307	1,035	1,765
Pooled estimate of cardiovascular hospital admissions			141	485	1,062

Exhibit A-7 Underlying Estimates and Weights for Pooled Estimate of Ozone-Related Asthma ER Visits

Study	Ages Affected	Study Weights	5th %ile	mean	95th %ile
Cody et al. (1992)	>26	0.49	139	345	540
Weisel et al. (1995)	>26	0.49	545	754	950
Stieb et al. (1996)	>29	0.02	375	2,931	5,341
Pooled estimate of asthma ER			109	346	551

Exhibit A-8 Underlying Estimates and Weights for Pooled Estimate of PM-Related Chronic Bronchitis Studies

Study	Ages Affected	Study Weights	5th %ile	mean	95th %ile
Abbey et al. (1993)	>26	0.32	275	2,025	3,670
Abbey et al. (1995b)	>26	0.16	356	2,819	5,134
Schwartz (1993)	>29	0.52	929	2,310	3,609
Pooled estimate of chronic bronchitis			610	2,296	4,066

Exhibit A-9 Underlying Estimates and Weights for Pooled Estimate of PM-related MRAD and Any-of-19 Studies

Study	Ages Affected	Study Weights	5th %ile	mean	95th %ile
Krupnick et al. (1990)	18-65	0.004	1,808,712	10,720,334	19,313,814
Ostro and Rothschild (1989b)	18-65	0.996	3,066,913	3,615,693	4,177,213
Pooled estimate of MRAD Any of 19			3,034,085	3,628,527	4,177,213

Exhibit A-10 Underlying Estimates and Weights for Pooled Estimate of Ozone-related MRAD and Any-of-19 Studies

Study	Ages Affected	Study Weights	5th %ile	mean	95th %ile
Krupnick et al. (1990)	18-65	0.04	821,285	6,019,281	11,145,526
Ostro and Rothschild (1989b)	18-65	0.96	1,014,435	2,061,615	3,118,562
Pooled estimate of MRAD Any of 19			1,014,435	2,226,463	3,414,837

APPENDIX B: OZONE CONCENTRATION-RESPONSE FUNCTIONS

Note that ΔO_3 is defined as ($O_{3,\ baseline}$ - $O_{3,\ control}$), and that the change is defined as: ($incidence_{control}$ - $incidence_{baseline}$).

B.1 SHORT-TERM OZONE-RELATED MORTALITY (FOUR U.S. STUDIES)

Four studies were used to estimate the possible relationship between ozone and increased mortality.

B.1.1 Short-Term Mortality (U.S.) (Ito et al., 1996)

Ito and Thurston (1996) examined the relationship between daily non-accidental mortality and air pollution levels in Cook County, Illinois from 1985 to 1990. They examined daily levels of ozone, PM_{10}, SO_2, and CO, and found a significant relationship for ozone and PM_{10} with both pollutants in the model; no significant effects were found for SO_2 and CO. The ozone coefficient is estimated from a model with PM_{10}.

The C-R function to estimate the change in short-term mortality associated with a change in ozone is:

$$\Delta Nonaccidental\ Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = county-level daily incidence for non-accidental deaths per person of any age
β = ozone coefficient = 0.000634 (Ito, 1998)[53]
ΔO_3 = change in daily one-hour maximum ozone concentration (ppb)
pop = population of all ages
$_{\beta}$ = standard error of β = 0.000251 (Ito, 1998).

Incidence Rate. To estimate county-specific baseline mortality incidence, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999).

[53]The published paper has an incorrect coefficient and standard error; updated estimates were obtained from the author.

B.1.2 Short-Term Mortality (U.S.) (Kinney et al., 1995)

Kinney et al. (1995) examined the relationship between daily non-accidental mortality and air pollution levels in Los Angeles, California from 1985 to 1990. They examined ozone, PM_{10}, and CO, and found a significant relationship for each pollutant in single pollutant models. The effect for ozone dropped to zero with the inclusion of PM_{10} in the model, while the effect for CO and PM_{10} appeared independent of each other and were of a similar magnitude.

The C-R function to estimate the change in short-term mortality associated with a change in ozone is:

$$\Delta Nonaccidental\ Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = county-level daily incidence for non-accidental deaths per person of any age
β = ozone coefficient = 0
ΔO_3 = change in daily 1-hour maximum ozone concentration (ppb)
pop = population of all ages
$_\beta$ = standard error of β = 0.000214

Incidence Rate. To estimate county-specific baseline mortality incidence, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999).

Coefficient Estimate (β). In a model with PM_{10}, the ozone coefficient (β) for non-accidental mortality is estimated from the relative risk (1.00) associated with a change in daily one-hour maximum ozone of 143 ppb (Kinney et al., 1995, Table 2 and Figure 3):

$$b = \frac{\ln(1.00)}{(143)} = 0.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Kinney et al., 1995, Table 2 and Figure 3):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.06)}{143} - \frac{\ln(1.00)}{143}\right)}{1.96} = 0.000208$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.00)}{143} - \frac{\ln(0.94)}{143}\right)}{1.96} = 0.000221$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000214.$$

B.1.3 Short-Term Mortality (U.S.) (Moolgavkar et al., 1995)

Moolgavkar et al. (1995) examined the relationship between daily non-accidental mortality and air pollution levels in Philadelphia, Pennsylvania from 1973 to 1988. They examined ozone, TSP, and SO_2 in a three-pollutant model, and found a significant relationship for ozone and SO_2; TSP was not significant.

The C-R function to estimate the change in short-term mortality associated with a change in ozone is:

$$\Delta Nonaccidental\ Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = county-level daily incidence for non-accidental deaths per person of any age
β = ozone coefficient = 0.000611
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages
$_\beta$ = standard error of β = 0.000216

Incidence Rate. To estimate county-specific baseline mortality incidence, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999).

Coefficient Estimate (β). Based on a model with TSP and SO_2, the coefficient (β) for non-accidental mortality is estimated from the relative risk (1.063) associated with a change in daily average ozone of 100 ppb (Moolgavkar et al., 1995, Table 5):

$$b = \frac{\ln(1.063)}{(100)} = 0.000611.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Moolgavkar et al., 1995, Table 5):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.108)}{100} - \frac{\ln(1.063)}{100}\right)}{1.96} = 0.000212$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.063)}{100} - \frac{\ln(1.018)}{100}\right)}{1.96} = 0.000221$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000216.$$

B.1.4 Short-Term Mortality (U.S.) (Samet et al., 1997)

Samet et al. (1997) examined the relationship between daily non-accidental mortality and air pollution levels in Philadelphia, Pennsylvania from 1974 to 1988. They examined ozone, TSP, SO_2, NO_2, and CO in a five-pollutant model, and found a significant relationship for each pollutant.

The C-R function to estimate the change in short-term mortality associated with a change in ozone is:

$$\Delta Nonaccidental\ Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = county-level daily incidence for non-accidental deaths per person of any age
β = ozone coefficient = 0.000936
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages
$_\beta$ = standard error of β = 0.000312

Incidence Rate. To estimate county-specific baseline mortality incidence, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999).

Coefficient Estimate (β). In a model with TSP, SO_2, NO_2, and CO, the ozone coefficient (β) for non-accidental mortality is estimated from the relative risk (1.0191) associated with a change in the two-day average ozone level of 20.219 ppb (Samet et al., 1997, Table 9):

$$b = \frac{\ln(1.0191)}{(20.219)} = 0.000936.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated using the reported t-value (t=3) (Samet et al., 1997, Table 9):

$$s_b = \frac{.000936}{3} = 0.000312.$$

B.2 CHRONIC ILLNESS

= population of In recent years, a number of studies have investigated the possible link between ozone and the development of chronic illness. Abbey et al. (1991; 1993) reported a significant link between ozone and the development of asthma, and Portney and Mullahy (1990) found ozone linked to sinusitis and hay fever. A review of research data by EPA (1996a, p. 9-35) concluded that prolonged ozone exposure causes structural changes in several regions of the respiratory tract, and the available epidemiological studies are suggestive of a link between chronic health effects in humans and long-term ozone exposure. Most recently, a study by McDonnell et al. (1999) carefully measured ozone exposure for Seventh Day Adventists living in California.

B.2.1 Asthma Adult Onset (McDonnell et al., 1999)

The McDonnell et al. (1999) study used the same cohort of Seventh-Day Adventists as Abbey et al. (1991; 1993), and examined the association between air pollution and the onset of asthma in adults between 1977 and 1992. Males who did not report doctor-diagnosed asthma in 1977, but reported it in 1987 or 1992, had significantly higher ozone exposures, controlling for other covariates; no significant effect was found between ozone exposure and asthma in females. No significant effect was reported for females or males due to exposure to PM, NO_2, SO_2, or SO_4.

The C-R function to estimate the change in chronic asthma is:

$$\Delta Chronic\ Asthma = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta O_3 \cdot b} + y_0} - y_0\right] \cdot pop\,,$$

where:

y_0 = annual asthma incidence rate per person (McDonnell et al., 1999, Table 4) = 0.00219
β = estimated O_3 coefficient (McDonnell et al., 1999, Table 5) = 0.0277
ΔO_3 = change in annual average 8-hour O_3 concentration[54]
pop = population of non-asthmatic males ages 27 and older[55] = 96.66% of males 27+
$_{\beta}$ = standard error of β (McDonnell et al., 1999, Table 5) = 0.0135

Incidence Rate. The annual incidence rate is derived by taking the number of new cases (32), dividing by the number of individuals in the sample (972), as reported by (McDonnell et al., 1999, Table 4), and then dividing by the 15 years in the sample.

[54]The eight-hour ozone concentration is defined as 9:00 A.M. to 4:59 P.M. The study used the 1973-1992 mean 8-hour average ambient ozone concentration (McDonnell et al., 1999, p. 113).

[55]The population weighted average incidence of asthma in males 27 and older is 3.34 percent. Population data from U.S. Census Bureau (1997, Table 14); asthma prevalence for males from Collins (1997, Table 9).

B.3 HOSPITAL ADMISSIONS

B.3.1 Hospital Admissions for Asthma (Burnett et al., 1999, Toronto)

Burnett et al. (1999) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada from1980 to 1994. They estimated multiple pollutant models, where pollutants for best fitting model were chosen using stepwise regression based on AIC criterion. Asthma admissions were linked to O_3, CO, and $PM_{2.5-10}$. This C-R function is based on the results of this three-pollutant model.

The C-R function to estimate the change in hospital admissions for asthma associated with daily changes in ozone is:

$$\Delta Asthma\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for asthma per person = 4.75 E-6
β = ozone coefficient = 0.00250
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages
$_\beta$ = standard error of β = 0.000718

Incidence Rate. Hospital admissions for obstructive lung disease (ICD-9 codes: 490-492, 496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.547 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 4.99 percent increase in admissions due to a ozone change of 19.5 ppb (Burnett et al., 1999, Tables 1 and 5). This translates to a relative risk of 1.0499. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0499)}{19.5} = 0.00250.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated using the t-value (t=3.48) (Burnett, 1999):

$$s_b = \frac{0.00250}{3.48} = 0.000718.$$

B.3.2 Hospital Admissions for Obstructive Lung Disease (Burnett et al., 1999, Toronto)

Burnett et al. (1999) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada from1980 to 1994. They estimated multiple pollutant models, where pollutants for the best fitting model were chosen using stepwise regression based on AIC criterion. Admissions for chronic obstructive pulmonary disease (COPD) were linked to O_3 and $PM_{2.5-10}$. This C-R function is based on the results of this two-pollutant model.

The C-R function to estimate the change in hospital admissions for obstructive lung disease associated with daily changes in ozone is:

$$\Delta \textit{Obstructive Lung Disease Admissions} = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for obstructive lung disease per person = 5.76 E-6
β = ozone coefficient = 0.00303
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages
$_\beta$ = standard error of β = 0.00110

Incidence Rate. Hospital admissions for respiratory infection (ICD-9 codes: 464, 466, 480-487, 494) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (1.485 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 6.08 percent increase in admissions due to a ozone change of 19.5 ppb (Burnett et al., 1999, Tables 1 and 5). This translates to a relative risk of 1.0608. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0608)}{19.5} = 0.00303.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated using the t-value (t=2.74) (Burnett, 1999):

$$s_b = \frac{0.00303}{2.74} = 0.00110.$$

B.3.3 Hospital Admissions for Respiratory Infection (Burnett et al., 1999, Toronto)

Burnett et al. (1999) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada from 1980 to 1994. They estimated multiple pollutant models, where pollutants for the best fitting model were chosen using stepwise regression based on AIC criterion. Respiratory infection admissions were linked to O_3, NO_2, and $PM_{2.5}$. This C-R function is based on the results from this three-pollutant model.

The C-R function to estimate the change in hospital admissions for respiratory infection associated with daily changes in ozone is:

$$\Delta\ Respiratory\ Infection\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for respiratory infection per person = 1.56 E-5
β = ozone coefficient = 0.00198
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages
$_\beta$ = standard error of β = 0.000520

Incidence Rate. Hospital admissions for respiratory infections (ICD-9 codes: 464-466, 480-486, 490-494, 496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (2.452 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 3.93 percent increase in admissions due to a ozone change of 19.5 ppb (Burnett et al., 1999, Tables 1 and 5). This translates to a relative risk of 1.0393. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0393)}{19.5} = 0.00198\ .$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated using the t-value (t=3.80) (Burnett, 1999):

$$s_b = \frac{0.00198}{3.80} = 0.000520\ .$$

B.3.4 Hospital Admissions for All Respiratory (Burnett et al., 1997, Toronto)

Burnett et al. (1997) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada during the summers of 1992-1994. All respiratory admissions were linked to coefficient of haze (COH) and O_3; other PM measures were less strongly linked. In two pollutant models, they found that CO, NO_2, and SO_2 were not significant, controlling for COH. They found that O_3 was still significant, controlling for COH. This C-R function is based on the results from the four-pollutant model ($PM_{2.5-10}$, O_3, NO_2, and SO_2) to estimate all respiratory incidence.

The C-R function to estimate the change in all respiratory hospital admissions associated with daily changes in ozone is:

$$\Delta All\ Respiratory\ Admissions = -\left[y_0 \cdot \left(e^{-b \cdot \Delta O_3} - 1\right)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for all respiratory admissions per person = 2.58 E-5
β = O_3 coefficient = 0.00498
ΔO_3 = change in daily 12-hour average O_3 concentration (ppb)[56]
pop = population of all ages
$_\beta$ = standard error of β = 0.00106

Incidence Rate. Hospital admissions for all respiratory causes (ICD-9 codes: 464-466, 480-486, 490-494, 496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (2.452 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a relative risk of 1.059 due to a change of 11.50 ppb in the daily average for O_3 (Burnett et al., 1997, Tables 2 and 6). The coefficient is calculated as follows:

$$b = \frac{\ln(1.059)}{11.50} = 0.00498.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated using the t-value (t=4.71) (Burnett et al., 1997, Table 6)

$$s_b = \frac{.00498}{4.71} = 0.00106.$$

[56] Burnett et al. (1997, Table 2 and p. 614) reported using the daytime average ozone level from 8 A.M. to 8 P.M.

B.3.5 Hospital Admissions for All Respiratory (Thurston et al., 1994, Toronto)

Thurston et al. (1994) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada, for six weeks in July and August 1986-1988. In single-pollutant models, ozone and various measures of PM were linked to all respiratory admissions. In two-pollutant models, ozone was still significant, but measures of PM were often not significant; only H^+ was significant. This C-R function is based on the results of a two-pollutant model ($PM_{2.5}$ and ozone).

The C-R function to estimate the change in all respiratory hospital admissions associated with daily changes in ozone is:

$$\Delta \, All\,Respiratory\,Admissions = b \cdot \Delta O_3 \cdot pop\,,$$

where:

β = ozone coefficient = 1.68 E-8
ΔO_3 = change in daily one-hour maximum ozone concentration (ppb)
pop = population of all ages
$_{\beta}$ = standard error of β = 9.71 E-9 .

Coefficient Estimate (β). Based on a linear model with $PM_{2.5}$, the one-hour maximum ozone coefficient comes from an estimated coefficient of 0.0404, which estimates admissions per ppb of ozone (Thurston et al., 1994, Table 3).[57] The population of Toronto was estimated to be 2.4 million (U.S. EPA, 1997a, Table D-7). We estimated a coefficient estimating admissions per person per ppb of ozone as follows:

$$b = \frac{0.0404}{2{,}400{,}000} = 1.68E-8.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated in a similar fashion (Thurston et al., 1994, Table 3):

$$s_b = \frac{0.0233}{2{,}400{,}000} = 9.71E-9.$$

[57]The 812 Retrospective analysis (U.S. EPA, 1997a, Table D-7) used an ozone coefficient based on a model with PM_{10}.

B.3.6 Hospital Admissions for Pneumonia (Moolgavkar et al., 1997, Minneapolis)

Moolgavkar et al. (1997) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1991. In a four pollutant model examining pneumonia admissions in Minneapolis, ozone was significant, while NO_2, SO_2, and PM_{10} were not significant. This C-R function is based on the results from the four-pollutant model to estimate pneumonia incidence.

The C-R function to estimate the change in hospital admissions for pneumonia associated with daily changes in ozone is:

$$\Delta Pneumonia\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for pneumonia per person = 5.30 E-5
β = O_3 coefficient = 0.00370
ΔO_3 = change in daily average O_3 concentration (ppb)
pop = population of ages 65 and older
$_\beta$ = standard error of β = 0.00103

Incidence Rate. Hospital admissions for pneumonia (ICD-9 codes: 480-487) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.642 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 5.7 percent increase in admissions due to a O_3 change of 15 ppb (Moolgavkar et al., 1997, Table 4 and p. 366); the model with a 130 df smoother was reported to be optimal (p. 368). This translates to a relative risk of 1.057. The coefficient is calculated as follows:

$$b = \frac{\ln(1.057)}{15} = 0.00370.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Moolgavkar et al., 1997, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.089)}{15} - \frac{\ln(1.057)}{15}\right)}{1.96} = 0.00101$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.057)}{15} - \frac{\ln(1.025)}{15}\right)}{1.96} = 0.00105$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00103.$$

B.3.7 Hospital Admissions for COPD (Moolgavkar et al., 1997, Minneapolis)

Moolgavkar et al. (1997) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1991. No significant effect found for any pollutant; the effect for ozone was marginally significant. This C-R function is based on the results from a three-pollutant model (O_3, CO, PM_{10}) to estimate COPD incidence.

The C-R function to estimate the change in hospital admissions for COPD associated with daily changes in ozone is:

$$\Delta\, COPD\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for COPD per person = 3.75 E-5
β = O_3 coefficient = 0.00274
ΔO_3 = change in daily average O_3 concentration (ppb)
pop = population of ages 65 and older
$_\beta$ = standard error of β = 0.00170

Incidence Rate. Hospital admissions for COPD (ICD-9 codes: 490-496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.454 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 4.2 percent increase in admissions due to a O_3 change of 15 ppb (Moolgavkar et al., 1997, Table 4 and p. 366); the model with a 100 df smoother was reported to be optimal (p. 368). This translates to a relative risk of 1.042. The coefficient is calculated as follows:

$$b = \frac{\ln(1.042)}{15} = 0.00274.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Moolgavkar et al., 1997, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.094)}{15} - \frac{\ln(1.042)}{15}\right)}{1.96} = 0.00166$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.042)}{15} - \frac{\ln(0.99)}{15}\right)}{1.96} = 0.00174$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00170.$$

B.3.8 Hospital Admissions for Pneumonia (Schwartz, 1994c, Minneapolis)

Schwartz (1994c) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1989. In a two-pollutant model, Schwartz found PM_{10} significantly related to pneumonia; ozone was weakly linked to pneumonia. This C-R function is based on the results of the two-pollutant model (PM_{10}, O_3) to estimate pneumonia incidence.

The C-R function to estimate the change in hospital admissions for pneumonia associated with daily changes in ozone is:

$$\Delta Pneumonia\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for pneumonia per person = 5.30 E-5
β = O_3 coefficient = 0.00280
ΔO_3 = change in daily average O_3 concentration (ppb)
pop = population of ages 65 and older
$_\beta$ = standard error of β = 0.00172

Incidence Rate. Hospital admissions for pneumonia (ICD-9 codes: 480-487) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.642 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with ozone, the coefficient (β) is estimated from the relative risk (1.15) associated with a 50 ppb change in the daily average ozone level (Schwartz, 1994c, Table 4 and p. 369):

$$b = \frac{\ln(1.15)}{50} = 0.00280.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1994c, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.36)}{50} - \frac{\ln(1.15)}{50}\right)}{1.96} = 0.00171$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.15)}{50} - \frac{\ln(0.97)}{50}\right)}{1.96} = 0.00174$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00172 .$$

B.3.9 Hospital Admissions for Pneumonia (Schwartz, 1994b, Detroit)

Schwartz (1994b) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Detroit, Michigan, from January 1986 to December 1989. In a two-pollutant model, Schwartz found both PM_{10} and ozone significantly linked to pneumonia and COPD; no significant link to asthma admissions was found for either pollutant. We use the results of this two-pollutant model.

The C-R function to estimate the change in hospital admissions for pneumonia associated with daily changes in ozone is:

$$\Delta Pneumonia\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop ,$$

where:

y_0 = daily hospital admission rate for pneumonia per person = 5.18 E-5
β = O_3 coefficient (Schwartz, 1994b, Table 4) = 0.00521
ΔO_3 = change in daily average O_3 concentration (ppb)
pop = population of ages 65 and older
$_\beta$ = standard error of β (Schwartz, 1994b, Table 4) = 0.0013

Incidence Rate. Hospital admissions for pneumonia (ICD-9 codes: 480-486) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.627 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

B.3.10 Hospital Admissions for COPD (Schwartz, 1994b, Detroit)

Schwartz (1994b) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Detroit, Michigan, from January 1986 to December 1989. In a two-pollutant model, Schwartz found both PM_{10} and ozone significantly linked to pneumonia and COPD; no significant link to asthma admissions was found for either pollutant. We use the results of this two-pollutant model.

The C-R function to estimate the change in hospital admissions for COPD associated with daily changes in O_3 is:

$$\Delta\, COPD\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for COPD per person = 3.05 E-5
β = O_3 coefficient (Schwartz, 1994b, Table 4) = 0.00549
ΔO_3 = change in daily average O_3 concentration
pop = population of ages 65 and older
$_{\beta}$ = standard error of β (Schwartz, 1994b, Table 4) = 0.00205

Incidence Rate. Hospital admissions for COPD (ICD-9 codes: 491-492, 494-496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.369 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

B.3.11 Hospital Admissions for All Respiratory (Schwartz, 1995, New Haven)

Schwartz (1996) examined the relationship between air pollution and hospital admissions for individuals 65 and older in New Haven, Connecticut, from January 1988 to December 1990. In single-pollutant models, PM_{10} and SO_2 were significant, while ozone was marginally significant. In two-pollutant models, ozone was significant in one of two models, and had stable coefficient estimates; PM_{10} was significant in two of two models, but had less stable estimates. SO_2 was significant in one of four models. The C-R function in this analysis is based on a two-pollutant model with ozone and PM_{10}.

The C-R function to estimate the change in all respiratory hospital admissions associated with daily changes in ozone is:

$$\Delta \, All\ Respiratory\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admissions for all respiratory conditions per person 65 and older = 1.187 E-4
β = ozone coefficient = 0.00265
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of ages 65 and older
$_{\beta}$ = standard error of β = 0.00140

Incidence Rate. All respiratory hospital admissions (ICD-9 codes: 460-519) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the national annual number of first-listed diagnoses for discharges (1.437 million) divided by the 1994 U.S. population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with PM_{10}, the coefficient (β) is estimated from the relative risk (1.07) associated with a change in ozone exposure of 50 $\mu g/m^3$ (Schwartz, 1995, Table 3 and p. 535):[58]

$$b = \frac{\ln(1.07)}{\left(\frac{50}{1.96}\right)} = 0.00265.$$

[58]A conversion of 1.96 $\mu g/m^3$ per ppb is used, based on a density of ozone of 1.96 grams per liter (at 25 degrees Celsius). Since there are 1000 liters in a cubic meter and a million µg in a gram, this density means that there are 1.96 billion µg of ozone in a cubic meter of ozone. If a cubic meter has just one ppb of ozone, then this means that this particular cubic meter has 1.96 µg of ozone (i.e., one ppb = 1.96 $\mu g/m^3$).

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1995, Table 3).

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.15)}{50/1.96} - \frac{\ln(1.07)}{50/1.96}\right)}{1.96} = 0.00144$$

$$s_{b,low} = \frac{b - b_{low} -}{1.96} = \frac{\left(\frac{\ln(1.07)}{50/1.96} - \frac{\ln(1.00)}{50/1.96}\right)}{1.96} = 0.00135$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00140.$$

B.3.12 Hospital Admissions for All Respiratory (Schwartz, 1995, Tacoma)

Schwartz (1996) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Tacoma, Washington, from January 1988 to December 1990. In single-pollutant models, PM_{10}, ozone, and SO_2 were all significant. In two-pollutant models, ozone was significant in two of two models, and had stable coefficient estimates; PM_{10} was significant in one of two models, but had less stable estimates; SO_2 was not significant in either of the two-pollutant models. The C-R function in this analysis is based on a two-pollutant model with ozone and PM_{10}.

The C-R function to estimate the change in hospital admissions for all-respiratory causes associated with daily changes in ozone is:

$$\Delta \, All\ Respiratory\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admissions for all respiratory conditions per person 65 and older = 1.187 E-4
β = ozone coefficient = 0.00715
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of ages 65 and older
$_{\beta}$ = standard error of β = 0.00257

Incidence Rate. All respiratory hospital admissions (ICD-9 codes: 460-519) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the national annual number of first-listed diagnoses for discharges (1.437 million) divided by the 1994 U.S. population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with PM_{10}, the coefficient (β) is estimated from the relative risk (1.20) associated with a change in ozone exposure of 50 $\mu g/m^3$ (Schwartz, 1995, Table 6 and p. 535):[59]

$$b = \frac{\ln(1.20)}{\left(\frac{50}{1.96}\right)} = 0.00715.$$

[59]A conversion of 1.96 $\mu g/m^3$ per ppb is used, based on a density of ozone of 1.96 grams per liter (at 25 degrees Celsius). Since there are 1000 liters in a cubic meter and a million µg in a gram, this density means that there are 1.96 billion µg of ozone in a cubic meter of ozone. If a cubic meter has just one ppb of ozone, then this means that this particular cubic meter has 1.96 µg of ozone (i.e., one ppb = 1.96 $\mu g/m^3$).

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1995, Table 6):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.37)}{50/1.96} - \frac{\ln(1.20)}{50/1.96}\right)}{1.96} = 0.00265$$

$$s_{b,low} = \frac{b - b_{low} -}{1.96} = \frac{\left(\frac{\ln(1.20)}{50/1.96} - \frac{\ln(1.06)}{50/1.96}\right)}{1.96} = 0.00248$$

$$s_{b} = \frac{s_{high} + s_{low}}{2} = 0.00257 .$$

B.3.13 Hospital Admissions for Cardiac (Burnett et al., 1997, Toronto)

Burnett et al. (1997) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada during the summers of 1992-1994. COH and ozone were significantly linked to cardiac admissions; other PM measures less strongly linked. In two-pollutant models, they found CO, NO_2, and SO_2 were not significant, when controlling for COH. Ozone was significant, controlling for COH. In four-pollutant models, COH and O_3 were both significant; no effect for NO_2 and SO_2. The C-R function in this analysis is based on a two-pollutant model with ozone and $PM_{2.5-10}$.

The C-R function to estimate the change in cardiac hospital admissions associated with daily changes in ozone is:

$$\Delta Cardiac\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for cardiac problems per person = 3.81 E-5
β = O_3 coefficient = 0.00531
ΔO_3 = change in daily 12-hour average O_3 concentration (ppb)[60]
pop = population of all ages
$_\beta$ = standard error of β = 0.00142

Incidence Rate. Hospital admissions for cardiac (ICD-9 codes: 410-414, 427-428) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (3.617 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a relative risk of 1.063 due to a O_3 change of 11.50 ppb (Burnett et al., 1997, Tables 2 and 5). The coefficient is calculated as follows:

$$b = \frac{\ln(1.063)}{11.50} = 0.00531.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated using the t-value (t=3.74) (Burnett et al., 1997, Table 5)

$$s_b = \frac{.00531}{3.74} = 0.00142.$$

[60] Burnett et al. (1997, Table 2 and p. 614) reported using the daytime average ozone level from 8 A.M. to 8 P.M.

B.3.14 Hospital Admissions for Dysrhythmias (Burnett et al., 1999, Toronto)

Burnett et al. (1999) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada from1980 to 1994. They estimated multiple pollutant models, where pollutants for best fitting model were chosen using stepwise regression based on AIC criterion. Dysrhythmias admissions were linked to O_3, CO, and $PM_{2.5}$. This C-R function is based on the results of this three-pollutant model.

The C-R function to estimate the change in hospital admissions for dysrhythmias associated with daily changes in ozone is:

$$\Delta Dysrhythmias\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for dysrhythmias per person = 6.46 E-6
β = ozone coefficient = 0.00168
ΔO_3 = change in daily average ozone concentration (ppb)
pop = population of all ages
$_{\beta}$ = standard error of β = 0.00103

Incidence Rate. Hospital admissions for dysrhthmias (ICD-9 code: 427) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.614 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 3.34 percent increase in admissions due to a ozone change of 19.5 ppb (Burnett et al., 1999, Tables 1 and 5). This translates to a relative risk of 1.0334. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0334)}{19.5} = 0.00168.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated using the t-value (t=1.63) (Burnett, 1999):

$$s_b = \frac{0.00168}{1.63} = 0.00103.$$

B.4 EMERGENCY ROOM VISITS

There is a wealth of epidemiological information on the relationship between air pollution and hospital admissions for various respiratory and cardiovascular diseases; in addition, some studies have examined the relationship between air pollution and ER visits. Because most ER visits do not result in an admission to the hospital -- the majority of people going to the ER are treated and return home -- we treat hospital admissions and ER visits separately, taking account of the fraction of ER visits that do get admitted to the hospital, as discussed below.

The only types of ER visits that have been explicitly linked to ozone in U.S. and Canadian epidemiological studies are asthma visits. However, it seems likely that ozone may be linked to other types of respiratory-related ER visits.

B.4.1 Emergency Room Visits for Asthma (Cody et al., 1992, Northern NJ)

Cody et al. (1992) examined the relationship between ER visits and air pollution for persons of all ages in central and northern New Jersey, from May to August in 1988-1989. In a two pollutant model, ozone was linked to asthma visits, and no effect was seen for SO_2. PM_{10} considered in separate analysis, because of limited (every sixth day) sampling; no significant effect was seen for PM_{10}.

The C-R function to estimate the change in asthma ER visits associated with daily changes in ozone is:

$$\Delta \, Asthma\ ERVisits = \frac{b}{BasePop} \cdot \Delta O_3 \cdot pop \cdot (1 - 0.37),$$

where:

β = ozone coefficient (Cody et al., 1992, Table 6) = 0.0203
BasePop = baseline population in northern New Jersey[61] = 4,436,976
ΔO_3 = change in daily five-hour average ozone concentration (ppb)[62]
pop = population of all ages
$_{\beta}$ = standard error of β (Cody et al., 1992, Table 6) = 0.00717

Correction for Double Counting. Smith et al. (1997, p. 789) reported that in 1987 there were 445,000 asthma admissions and 1.2 million asthma ER visits. Assuming that all asthma hospital admissions pass through the ER room, then 37% of ER visits end up as hospital admissions. This percentage is then subtracted from the estimated change in asthma-related ER visits.

[61]The population estimate is based on the 1990 population for the eight counties containing hospitals or in the central core of the study. Cody et al. (1992, Figure 1) presented a map of the study area; the counties are: Bergen, Essex, Hudson, Middlesex, Morris, Passaic, Somerset, and Union.

[62]The coefficients in the study were based on the five-hour (10:00 am to 2:59 pm) ozone average in ppm; they have been converted to ppb.

B.4.2 Emergency Room Visits for Asthma (Weisel et al., 1995, Northern NJ)

Weisel et al. (1995) examined the relationship between ER visits and air pollution for persons of all ages in central and northern New Jersey, from May to August in 1986-1990. A significant relationship was reported for ozone.

The C-R function to estimate the change in asthma ER visits associated with daily changes in ozone is:

$$\Delta\, Asthma\ ERVisits = \frac{b}{BasePop} \cdot \Delta O_3 \cdot pop \cdot (1 - 0.37),$$

where:

β = ozone coefficient = 0.0443
BasePop = baseline population in northern New Jersey[63] = 4,436,976
ΔO_3 = change in daily five-hour average ozone concentration (ppb)[64]
pop = population of all ages
$_{\beta}$ = standard error of β = 0.00723

Correction for Double Counting. Smith et al. (1997, p. 789) reported that in 1987 there were 445,000 asthma admissions and 1.2 million asthma ER visits. Assuming that all asthma hospital admissions pass through the ER room, then 37% of ER visits end up as hospital admissions. This percentage is then subtracted from the estimated change in asthma-related ER visits.

Coefficient Estimate (β). The coefficient used in the C-R function is a weighted average of the coefficients in Weisel et al. (1995, Table 2) using the inverse of the variance as the weight:

$$b = \left(\frac{\sum_{i=1986}^{1990} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1986}^{1990} \frac{1}{s_{b_i}^2}} \right) = 0.0443.$$

Standard Error ($_{\beta}$). The standard error of the coefficient ($_{\beta}$) is calculated as follows, assuming that the estimated year-specific coefficients are independent:

[63]The population estimate is based on the 1990 population for the eight counties containing hospitals or in the central core of the study. Cody et al. (1992, Figure 1) presented a map of the study area; the counties are: Bergen, Essex, Hudson, Middlesex, Morris, Passaic, Somerset, and Union.

[64]The coefficients in the study were based on the five-hour (10:00 am to 2:59 pm) ozone average in ppm; they have been converted to ppb.

$$s_b^2 = \text{var}\left(\frac{\sum_{i=1986}^{1990} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1986}^{1990} \frac{1}{s_{b_i}^2}} \right) = \left(\frac{\sum_{i=1986}^{1990} \frac{b_i}{s_{b_i}^2}}{g} \right) = \sum_{i=1986}^{1990} \text{var}\left(\frac{b_i}{s_{b_i}^2 \cdot g} \right).$$

This eventually reduces down to:

$$s_b^2 = \frac{1}{g} \Rightarrow s_b = \sqrt{\frac{1}{g}} = 0.00723.$$

B.4.3 Emergency Room Visits for Asthma (Stieb et al., 1996, New Brunswick)

Stieb et al. (1996) examined the relationship between ER visits and air pollution for persons of all ages in St. John, New Brunswick, Canada, from May through September in 1984-1992. Ozone was significantly linked to ER visits, especially when ozone levels exceeded 75 ppb.

The C-R function to estimate the change in asthma ER visits associated with daily changes in ozone is:

$$\Delta\, Asthma\ ERVisits = \frac{b}{BasePop} \cdot \Delta O_3 \cdot pop \cdot (1 - 0.37),$$

where:

β	= ozone coefficient (Stieb et al., 1996, Table 2 linear model) = 0.0035
BasePop	= baseline population in Saint John, New Brunswick (Stieb et al., 1996, p. 1354) = 125,000
ΔO_3	= change in the daily one-hour maximum ozone concentration (ppb)
pop	= population of all ages
$_{\beta}$	= standard error of β (Stieb et al., 1996, Table 2 linear model) = 0.0018

Correction for Double Counting. Smith et al. (1997, p. 789) reported that in 1987 there were 445,000 asthma admissions and 1.2 million asthma ER visits. Assuming that all asthma hospital admissions pass through the ER room, then 37% of ER visits end up as hospital admissions. This percentage is then subtracted from the estimated change in asthma-related ER visits.

B.5 ACUTE MORBIDITY

B.5.1 Any of 19 Respiratory Symptoms: Krupnick (1990)

Krupnick et al. (1990) estimated the impact of air pollution on the incidence of any of 19 respiratory symptoms or conditions in 570 adults and 756 children living in three communities in Los Angeles, California from September 1978 to March 1979. Krupnick et al. (1990) listed 13 specific "symptoms or conditions": head cold, chest cold, sinus trouble, croup, cough with phlegm, sore throat, asthma, hay fever, doctor-diagnosed ear infection, flu, pneumonia, bronchitis, and bronchiolitis. The other six symptoms or conditions are not specified.

In their analysis, they included coefficient of haze (COH, a measure of particulate matter concentrations), ozone, NO_2, and SO_2, and they used a logistic regression model that takes into account whether a respondent was well or not the previous day. A key difference between this and the usual logistic model, is that the model they used includes a lagged value of the dependent variable. In single-pollutant models, daily O_3, COH, and SO_2 were significantly related to respiratory symptoms in adults. Controlling for other pollutants, they found that ozone was still significant. The results were more variable for COH and SO_2, perhaps due to collinearity. NO_2 had no significant effect. No effect was seen in children for any pollutant. The results from the two-pollutant model with COH and ozone are used to develop a C-R function.

The C-R function used to estimate the change in ARD2 associated with a change in daily one-hour maximum ozone is based on Krupnick et al. (1990, p. 12):[65]

$$\Delta ARD2 \cong b^{*} \cdot \Delta O_3 \cdot pop \ ,$$

where:

β^*	=	first derivative of the stationary probability = 0.000137
ΔO_3	=	change in daily one-hour maximum ozone concentration (ppb)[66]
pop	=	population aged 18-65 years old[67]
$_{\beta}$	=	standard error of β^* = 0.0000697

Coefficient Estimate (β^*). The logistic regression model used by Krupnick et al. (1990) takes into account whether a respondent was well or not the previous day. Following Krupnick et al. (p. 12), the probability that one is sick is on a given day is:

$$probability(ARD2) = \frac{p_0}{1 - p_1 + p_0}$$

[65]Krupnick and Kopp (1988, p. 2-24) and ESEERCO (1994, p. V-32) used the same C-R functional form as that used here.

[66]Krupnick et al. (1990) used parts per hundred million (pphm) to measure ozone; the coefficient used here is based on ppb.

[67]The coefficient estimates are based on the sample of "adults," and assumes that individuals 18 and older were considered adult. According to Krupnick et al. (1990, Table 1), about 0.6 percent of the study sample was over the age of 60. This is a relatively small fraction, so it is further assumed that the results do not apply to individuals over the age of 65.

$$p_i = probability(ARD2 | sickness or not_{t-1}) = \frac{1}{1 - e^{b_0 + b_1 \cdot ARD2_{t-1} + X \cdot b}}, for\ i = 0,1.$$

where:

X	=	the matrix of explanatory variables
p_0	=	the probability of sickness on day t, given wellness on day t-1, and
p_1	=	the probability of sickness on day t, given sickness on day t-1.

In other words, the transition probabilities are estimated using a logistic function; the key difference between this and the usual logistic model, is that the model includes a lagged value of the dependent variable.

To calculate the impact of ozone (or other pollutants) on the probability of ARD2, it is possible, in principle, to estimate ARD2 before the change in ozone and after the change:

$$\Delta ARD2 = ARD2_{after} - ARD2_{before}.$$

However the full suite of coefficient estimates are not available.[68] Rather than use the full suite of coefficient values, the impact of ozone on the probability of ARD2 may be approximated by the derivative of ARD2 with respect to ozone:[69]

$$\frac{\partial probability(ARD2)}{\partial O_3} = \frac{p_0 \cdot (1 - p_1) \cdot b \cdot [p_1 + (1 - p_0)]}{(1 - p_1 + p_0)^2} = b^*,$$

where β is the reported logistic regression coefficient for ozone. The change in the incidence of ARD2 associated with a given change in ozone is then estimated by:

$$\frac{\partial ARD2}{\partial O_3} \cong \frac{\Delta ARD2}{\Delta O_3}$$

$$\Rightarrow \frac{\Delta ARD2}{\Delta O_3} \cong b^*$$

$$\Rightarrow \Delta ARD2 \cong b^* \cdot \Delta O_3.$$

[68]The model without NO_2 (Krupnick et al., 1990, Table V equation 3) was used in this analysis, but the full suite of coefficient estimates for this model were not reported. Krupnick et al. (Table IV) reported all of the estimated coefficients for a model of children and for a model of adults when four pollutants were included (ozone, COH, SO_2, and NO_2). However, because of high collinearity between NO_2 and COH, NO_2 was dropped from some of the reported analyses (Krupnick et al., p. 10), and the resulting coefficient estimates changed substantially (see Krupnick et al., Table V). Both the ozone and COH coefficients dropped by about a factor of two or more.

[69]The derivative result is reported by Krupnick et al. (1990, p. 12).

This analysis uses transition probabilities obtained from Krupnick et al. as reported by ESEERCO (1994, p. V-32) for the adult population: $p_1 = 0.7775$ and $p_0 = 0.0468$. This implies:

$$b^* = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.00055 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 0.000137 .$$

Standard Error ($_\beta$). The standard error for the coefficient ($_\beta$) is derived using the reported standard error of the logistic regression coefficient in Krupnick et al. (1990, Table V):

$$b_{high} = 0.00055 + (1.96 \cdot 0.00027) = 0.00108$$

$$\Rightarrow b^*_{high} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.00108 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 0.000268$$

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{(0.000268 - 0.000137)}{1.96} = 0.0000668$$

$$b_{low} = 0.00055 - (1.96 \cdot 0.00027) = 0.0000208$$

$$\Rightarrow b^*_{low} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.0000208 \cdot [0.7775 + (1 - 0.0468)]}{(1 - 0.7775 + 0.0468)^2} = 5.17 \cdot 10^{-6}$$

$$\Rightarrow s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(0.000137 + 5.17 \cdot 10^{-6}\right)}{1.96} = 0.0000725$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.0000697 .$$

B.5.2 Minor Restricted Activity Days: Ostro and Rothschild (1989b)

Ostro and Rothschild (1989b) estimated the impact of $PM_{2.5}$ on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for $PM_{2.5}$, two-week average O_3 has highly variable association with RRADs and MRADs. Controlling for O_3, two-week average $PM_{2.5}$ was significantly linked to both health endpoints in most years.

The study is based on a "convenience" sample of individuals ages 18-65. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals 65 and younger. The elderly appear more likely to die due to PM exposure than other age groups (e.g., Schwartz, 1994d, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994a; Schwartz, 1994b).

Using the results of the two-pollutant model, we developed separate coefficients for each year in the analysis, which were then combined for use in this analysis. The coefficient used in this analysis is a weighted average of the coefficients (Ostro, 1987, Table IV) using the inverse of the variance as the weight. The C-R function to estimate the change in the number of minor restricted activity days (MRAD) associated with a change in daily O_3 is:

$$\Delta MRAD = -\left[y_0 \cdot (e^{-b \cdot \Delta O_3} - 1)\right] \cdot pop,$$

where:

y_0 = daily MRAD daily incidence rate per person = 0.02137
β = inverse-variance weighted O_3 coefficient = 0.00220
ΔO_3 = change in daily one-hour maximum ozone concentration (ppb)[70]
pop = adult population aged 18 to 65
$_{\beta}$ = standard error of β = 0.000658

Incidence Rate. The annual incidence rate (7.8) provided by Ostro and Rothschild (1989b, p. 243) was divided by 365 to get a daily rate of 0.02137.

Coefficient Estimate (β). The coefficient used in the C-R function is a weighted average of the coefficients in Ostro and Rothschild (1989b, Table 4) using the inverse of the variance as the weight:[71]

$$b = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = 0.00220.$$

[70]The study used a two-week average pollution concentration; the daily rate used here is assumed to be a reasonable approximation. The study used ozone measurements in $\mu g/m^3$; a conversion of 1.96 $\mu g/m^3$ = 1 ppb is assumed here.

[71]The calculation of the MRAD coefficient and its standard error is exactly analogous to the calculation done for the work-loss days coefficient based on Ostro (1987).

Standard Error ($_{\beta}$). The standard error of the coefficient ($_{\beta}$) is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$s_b^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}}\right) = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{g}\right) = \sum_{i=1976}^{1981} \text{var}\left(\frac{b_i}{s_{b_i}^2 \cdot g}\right).$$

This reduces down to:

$$s_b^2 = \frac{1}{g} \Rightarrow s_b = \sqrt{\frac{1}{g}} = 0.000658.$$

B.5.3 Asthma Attacks: Whittemore and Korn (1980)

Whittemore and Korn (1980) examined the relationship between air pollution and asthma attacks in a survey of 443 children and adults, living in six communities in southern California during three 34-week periods in 1972-1975. The analysis focused on TSP and oxidants (O_x). Respirable PM, NO_2, SO_2 were highly correlated with TSP and excluded from the analysis. In a two pollutant model, daily levels of both TSP and oxidants were significantly related to reported asthma attacks. The results from this model were used, and the oxidant result was adjusted below so it may be used with ozone data.

The C-R function to estimate the change in asthma attacks associated with a change in daily ozone is:

$$\Delta Asthma\ Attacks = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta O_3 \cdot b} + y_0} - y_0\right]\cdot pop,$$

where:

y_0 = daily incidence of asthma attacks = 0.027 (Krupnick, 1988, p. 4-6)
β = ozone coefficient = 0.00184
ΔO_3 = change in daily one-hour maximum ozone concentration (ppb)
pop = population of asthmatics of all ages = 5.61% of the population of all ages (Adams and Marano, 1995 Table 57).
$_{\beta}$ = standard error of β = 0.000714

Incidence Rate. The annual rate of 9.9 asthma attacks per astmatic is divided by 365 to get a daily rate. A figure of 9.9 is roughly consistent with the recent statement that "People with asthma have more than 100 million days of restricted activity" each year (National Heart, 1997). This 100 million incidence figure coupled with the 1996 population of 265,557,000 (U.S. Bureau of the Census, 1997, Table 2) and the latest asthmatic prevalence rate of 5.61% (Adams et al., 1995, Table 57), suggest an annual asthma attach rate per asthmatic of 6.7.

Coefficient Estimate (β). Based on a model with TSP, the daily one-hour ozone coefficient is based on an oxidant coefficient (1.66) estimated from data expressed in ppm (Whittemore et al., 1980, Table 5):[72]

$$b = \frac{1.66\cdot 1.11}{1000} = 0.00184.$$

[72]The study used oxidant measurements in ppm (Whittemore et al., 1980, p. 688); these have been converted to ozone measurements in ppb, assuming ozone comprises 90% of oxidants (i.e., 1.11*ozone=oxidant). It is assumed that the harm of oxidants is caused by ozone. The view expressed in the Ozone Staff Paper (U.S. EPA, 1996b, p.164) is consistent with assuming that ozone is the oxidant of concern at normal ambient concentrations: "Further, among the photochemical oxidants, the acute-exposure chamber, field, and epidemiological human health data base raises concern only for O_3 at levels of photochemical oxidants commonly reported in ambient air. Thus, the staff recommends that O_3 remain as the pollutant indicator for protection of public health from exposure to all photochemical oxidants found in the ambient air."

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) is calculated from the two-tailed p-value (<0.01) reported by Whittemore and Korn (1980, Table 5), which implies a t-value of at least 2.576 (assuming a large number of degrees of freedom).

$$s_b = \frac{b}{t} = \frac{0.184}{2.576} = 0.000714 .$$

B.5.4 Worker Productivity: Crocker and Horst (1981)

To monetize benefits associated with increased worker productivity resulting from improved ozone air quality, we used information reported in Crocker and Horst (1981) and summarized in EPA (1994). Crocker and Horst examined the impacts of ozone exposure on the productivity of outdoor citrus workers. The study measured productivity impacts as the change in income associated with a change in ozone exposure, given as the elasticity of income with respect to ozone concentration (-0.1427).[73] The reported elasticity translates a ten percent reduction in ozone to a 1.4 percent increase in income. Given the median daily income for outdoor workers engaged in strenuous activity reported by the 1990 U.S. Census, $89.64 per day (1997$), a ten percent reduction in ozone yields about $1.26 in increased daily wages. The median daily income for outdoor workers is a national estimate, however. We adjust this estimate to reflect regional variations in income using a factor based on the ratio of national median household income divided by a county's median household income. No information was available for quantifying the uncertainty associated with the central valuation estimate. Therefore, no uncertainty analysis was conducted for this endpoint.

[73] The relationship estimated by Crocker and Horst between wages and ozone is a log-log relationship. Therefore the elasticity of wages with respect to ozone is a constant, equal to the coefficient of the log of ozone in the model.

APPENDIX C: PARTICULATE MATTER C-R FUNCTIONS

Note that ΔPM is defined -- for all of the concentration-response (C-R) functions -- as $PM_{baseline}$ - $PM_{control}$, and that the change is defined to be: - ($incidence_{control}$ - $incidence_{baseline}$).

3.1 MORTALITY

There are two types of exposure to PM that may result in premature mortality. Short-term exposure may result in excess mortality on the same day or within a few days of exposure. Long-term exposure over, say, a year or more, may result in mortality in excess of what it would be if PM levels were generally lower, although the excess mortality that occurs will not necessarily be associated with any particular episode of elevated air pollution levels. In other words, long-term exposure may capture a facet of the association between PM and mortality that is not captured by short-term exposure.

3.1.1 Mortality (Pope et al., 1995)

Pope et al. (1995) used a Cox proportional hazard model to estimate the impact of long-term PM exposure. They followed 552,138 individuals ages 30 and over in 51 cities from September 1, 1982 to December 31, 1989, and related their survival to median $PM_{2.5}$ concentrations for 1979 to 1983. Pope et al. (1995, Table 2) reported results for all-cause deaths, lung cancer (ICD-9 code: 162), cardiopulmonary deaths (ICD-9 codes: 401-440 and 460-519), and "all other" deaths,[74] and found that median $PM_{2.5}$ is significantly related to all-cause and cardiopulmonary mortality. Pope et al. included only PM, so it is unclear to what extent it may be including the impacts of ozone or other gaseous pollutants.

Pope et al. (1995) is the better of the two published prospective cohort studies: it has a larger population and includes more cities than the prospective cohort study by Dockery et al. (1993). Pope et al.'s study has several further advantages. The population followed in this study was largely Caucasian and middle class, decreasing the likelihood that interlocational differences in premature mortality were due in part to differences in race, socioeconomic status, or related factors. In addition, the PM coefficient in Pope et al. is likely to be biased downward, counteracting a possible upward bias associated with historical air quality trends discussed earlier. One source of this downward bias is the generally healthier study population, in comparison to poorer minority populations. Another source of downward bias is that intercity movement of cohort members was not considered in this study. Migration across study cities would result in exposures of cohort members being more similar than would be indicated by assigning city-specific annual average pollution levels to each member of the cohort. The more intercity migration there is, the more exposure will tend toward an intercity mean. If this is ignored, differences in exposure levels, that are proxied by differences in city-specific annual average PM levels, will be exaggerated, and will result in a downward bias of the PM coefficient (because a given difference in mortality rates is being associated with a larger difference in PM levels than is actually the case).

[74] All-cause mortality includes accidents, suicides, homicides and legal interventions. The category "all other" deaths is all-cause mortality less lung cancer and cardiopulmonary deaths.

The C-R function to estimate the change in long-term mortality is:

$$\Delta Nonaccidental\ Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:
y_0 = county-level annual non-accidental death rate per person
β = $PM_{2.5}$ coefficient = 0.006408
$\Delta PM_{2.5}$ = change in annual median $PM_{2.5}$ concentration
pop = population of ages 30 and older
$_\beta$ = standard error of β = 0.001509

Incidence Rate. To estimate county-specific baseline mortality incidence among individuals ages 30 and over, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999). Note that Pope et al. (1995) used all cause mortality when estimating the impact of PM, however, it was decided to use non-accidental mortality in this analysis. Using non-accidental mortality (rather than all-cause mortality) underestimates deaths averted by about 7 percent; this is discussed in Abt Associates (1999, p. A-21).

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.17) associated with a change in median exposure going from 9 $\mu g/m^3$ to 33.5 $\mu g/m^3$ (Pope et al., 1995, Table 2).

$$b = \frac{\ln(1.17)}{(33.5 - 9)} = 0.006408.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Pope et al., 1995, Table 2).

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.26)}{24.5} - \frac{\ln(1.17)}{24.5}\right)}{1.96} = 0.001543$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.17)}{24.5} - \frac{\ln(1.09)}{24.5}\right)}{1.96} = 0.001475$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.001509.$$

3.1.2 Mortality (Dockery et al., 1993)

Dockery et al. (1993) examined the relationship between PM exposure and mortality in a cohort of 8,111 individuals aged 25 and older, living in six U.S. cities. They surveyed these individuals in 1974-1977 and followed their health status until 1991. While they used a smaller sample of individuals from fewer cities than the study by Pope et al., they used improved exposure estimates, a slightly broader study population (adults aged 25 and older), and a follow-up period nearly twice as long as that of Pope et al. (1995). Perhaps because of these differences, Dockery et al. study found a larger effect of PM on premature mortality than that found by Pope et al.

The C-R function to estimate the change in long-term mortality is:

$$\Delta Nonaccidental\ Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = county-level annual non-accidental death rate per person
β = $PM_{2.5}$ coefficient = 0.0124
$\Delta PM_{2.5}$ = change in annual mean $PM_{2.5}$ concentration
pop = population of ages 25 and older
$_\beta$ = standard error of β = 0.00423

Incidence Rate. To estimate county-specific baseline mortality incidence among individuals ages 25 and over, this analysis used the average annual county mortality rate from 1994 through 1996 (U.S. Centers for Disease Control, 1999). Dockery et al. (1993, p. 1754) appear to have used all-cause mortality when estimating the impact of PM, however, it was decided to use non-accidental mortality in this analysis. Using non-accidental mortality (rather than all-cause mortality) underestimates deaths averted by about 7 percent; this is discussed in Abt Associates (1999, p. A-21).

Coefficient Estimate (β). The coefficient (β) is estimated from the relative risk (1.26) associated with a change in mean exposure going from 11.0 $\mu g/m^3$ to 29.6 $\mu g/m^3$ (Dockery et al., 1993, Tables 1 and 5):

$$b = \frac{\ln(1.26)}{(29.6 - 11)} = 0.0124.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Dockery et al., 1993, Table 5):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.47)}{18.6} - \frac{\ln(1.26)}{18.6}\right)}{1.96} = 0.00423$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.26)}{18.6} - \frac{\ln(1.08)}{18.6}\right)}{1.96} = 0.00423$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00423.$$

3.1.3 Neonatal Mortality (Woodruff et al., 1997)

In a study of four million infants in 86 U.S. metropolitan areas conducted from 1989 to 1991, Woodruff et al. (1997) found a significant link between PM_{10} exposure in the first two months of an infant's life with the probability of dying between the ages of 28 days and 364 days. PM_{10} exposure was significant for all-cause mortality. PM_{10} was also significant for respiratory mortality in average birth-weight infants, but not low birth-weight infants.

In addition to the work by Woodruff et al., work in Mexico City (Loomis et al., 1999), the Czech Republic (Bobak and Leon, 1992), Sao Paulo (Pereira et al., 1998; Saldiva et al., 1994), and Beijing (Wang et al., 1997) provides additional evidence that particulate levels are significantly related to infant or child mortality, low birth weight or intrauterine mortality.

Conceptually, neonatal or child mortality could be added to the premature mortality predicted by Pope et al. (1995), because the Pope function covers only the population over 30 years old.[75] However, the EPA Science Advisory Board recently advised the Agency not to include post-neonatal mortality in this analysis because the study is of a new endpoint and the results have not been replicated in other studies (U.S. EPA, 1999b, p. 12). The estimated avoided incidences of neonatal mortality are estimated and presented as a sensitivity analysis, and are not included in the primary analysis.

The C-R function to estimate the change in infant mortality is:

$$\Delta Infant\ Mortality = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot b} + y_0} - y_0\right]\cdot pop,$$

where:

y_0 = county annual postneonatal[76] infant deaths per infant under the age of one
β = PM_{10} coefficient = 0.00392
ΔPM_{10} = change in annual average PM_{10} concentration[77]
pop = population of infants under one year old
$_{\beta}$ = standard error of β = 0.00122

Coefficient Estimate (β). The estimated logistic coefficient (β) is based on the odds ratio (= 1.04) associated with a 10 $\mu g/m^3$ change in PM_{10} (Woodruff et al., 1997, Table 3). The coefficient is calculated as follows:

$$b_{PM_{10}} = \frac{\ln(1.04)}{10} = 0.00392\ .$$

[75] Predicted neonatal mortality could not be added to the premature mortality predicted by the daily (short-term exposure) mortality studies, however, because these studies cover all ages.

[76] Post-neonatal refers to infants that are 28 days to 364 days old.

[77] Woodruff et al. (1997) used PM_{10} exposure in the first two months of an infant's life.

Standard Error ($_{\beta}$). The standard error for the coefficient is calculated as the average of the standard errors implied by the reported lower and upper bounds of the odds ratio (1.02 to 1.07) (Woodruff et al., 1997, Table 3). This reproduces both the lower and upper bounds of the odds ratio:

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.07)}{10} - \frac{\ln(1.04)}{10}\right)}{1.96} = 0.001451$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.04)}{10} - \frac{\ln(1.02)}{10}\right)}{1.96} = 0.000991$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.00122 .$$

3.1.4 Short-Term Mortality (Schwartz et al., 1996)

Schwartz et al. (1996) pooled the results from six cities in the U.S. and found a significant relationship between daily $PM_{2.5}$ concentration and non-accidental mortality.[78] Abt Associates Inc. (1996b, p. 52) used the six $PM_{2.5}$ relative risks reported by Schwartz et al. in a three-step procedure to estimate a pooled $PM_{2.5}$ coefficient and its standard error. The first step estimates a random-effects pooled estimate of β; the second step uses an "empirical Bayes" procedure to reestimate the β for each study as a weighted average of the β reported for that location and the random effects pooled estimate; the third step estimates the underlying distribution of β, and uses a Monte Carlo procedure to estimate the standard error (Abt Associates Inc., 1996a, p. 65).

The C-R function to estimate the change in mortality associated with daily changes in $PM_{2.5}$ is:

$$\Delta Nonaccidental\ Mortality = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = county-level daily incidence for non-accidental deaths per person of any age
β = $PM_{2.5}$ coefficient (Abt Associates Inc., 1996a, Exhibit 7.2) = 0.001433
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration
pop = population of all ages
$_{\beta}$ = standard error of β (Abt Associates Inc., 1996a, Exhibit 7.2) = 0.000129

[78]Schwartz et al. (1996, p. 929) defined non-accidental mortality as all-cause mortality less deaths due to accidents and other external causes (ICD-9 codes: 800-999). Other external causes includes suicide, homicide, and legal intervention (National Center for Health Statistics, 1994).

3.2 CHRONIC MORBIDITY

Schwartz (1993) and Abbey et al. (1995b; 1993) provide evidence that PM exposure over a number of years gives rise to the development of chronic bronchitis in the U.S., and a recent study by McDonnell et al. (1999) provides evidence that ozone exposure is linked to the development of asthma in adults. These results are consistent with research that has found chronic exposure to pollutants leads to declining pulmonary functioning (Abbey et al., 1998; Ackermann-Liebrich et al., 1997; Detels et al., 1991).[79]

We estimate the changes in the new cases of chronic bronchitis using the studies by Schwartz (1993), Abbey et al. (1993), and Abbey et al. (1995b). The Schwartz study is somewhat older and uses a cross-sectional design, however, it is based on a national sample, unlike the Abbey et al. studies which are based on a sample of California residents. We first pool the estimates from the two studies by Abbey et al. – since they are based on the same sample population and simply use different measures of PM – and then pool this estimate with that from Schwartz.

3.2.1 Chronic Bronchitis (Schwartz, 1993)

Schwartz (1993) examined survey data collected from 3,874 adults ranging in age from 30 to 74, and living in 53 urban areas in the U.S. The survey was conducted between 1974 and 1975, as part of the National Health and Nutrition Examination Survey, and is representative of the non-institutionalized U.S. population. Schwartz (1993, Table 3) reported chronic bronchitis prevalence rates in the study population by age, race, and gender. Non-white males under 52 years old had the lowest rate (1.7%) and white males 52 years and older had the highest rate (9.3%). The study examined the relationship between the prevalence of reported chronic bronchitis, asthma, shortness of breath (dyspnea) and respiratory illness[80], and the annual levels of TSP, collected in the year prior to the survey (TSP was the only pollutant examined in this study). TSP was significantly related to the prevalence of chronic bronchitis, and marginally significant for respiratory illness. No effect was found for asthma or dyspnea.

Schwartz (1993) examined the *prevalence* of chronic bronchitis, not its *incidence*. To use Schwartz's study and still estimate the change in incidence, there are at least two possible approaches. The first is to simply assume that it is appropriate to use the baseline *incidence* of chronic bronchitis in a C-R function with the estimated coefficient from Schwartz's study, to directly estimate the change in incidence. The second is to estimate the percentage change in the prevalence rate for chronic bronchitis using the estimated coefficient from Schwartz's study in a C-R function, and then to assume that this percentage change applies to a baseline incidence rate obtained from another source. (That is, if the prevalence declines by 25 percent with a drop in PM, then baseline incidence drops by 25 percent with the same drop in PM.) This analysis is using the latter approach, and estimates a percentage change in prevalence which is then applied to a baseline incidence rate.

[79] There are a limited number of studies that have estimated the impact of air pollution on chronic bronchitis. An important hindrance is the lack of health data and the associated air pollution levels over a number of years.

[80] Respiratory illness defined as a significant condition, coded by an examining physician as ICD-8 code 460-519.

The C-R function to estimate the change in chronic bronchitis is:

$$\Delta Chronic\ Bronchitis = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot b} + y_0} - y_0\right]\cdot\left[\frac{z_0}{y_0}\right]\cdot pop\ ,$$

where:

y_0 = national chronic bronchitis prevalence rate for individuals 18 and older (Adams et al., 1995, Table 62 and 78) = 0.0535

z_0 = annual bronchitis incidence rate per person (Abbey et al., 1993, Table 3) = 0.00378

β = estimated PM_{10} logistic regression coefficient = 0.0123

ΔPM_{10} = change in annual average PM_{10} concentration

pop = population of ages 30 and older without chronic bronchitis = 0.9465*population 30+

$_{\beta}$ = standard error of β = 0.00434 .

Prevalence Rate. The national chronic bronchitis prevalence rate was not available for individuals 30 and older. Instead, we used the prevalence rate for individuals 18 and older (Adams et al., 1995, Table 62 and 78). The 1994 national figures are the latest available, and are suggested here.

Incidence Rate. The annual incidence rate is derived by taking the number of new cases (234), dividing by the number of individuals in the sample (3,310), as reported by Abbey et al.(1993, Table 3), dividing by the ten years covered in the sample, and then multiplying by one minus the reversal rate (the percentage of reversals is estimated to be 46.6% based on Abbey et al. (1995a, Table 1)). Using the same data base, Abbey et al. (1995a, Table 1) reported the incidences by three age groups (25-54, 55-74, and 75+) for "cough type" and "sputum type" bronchitis, but they did not report an overall incidence rate for bronchitis.

Coefficient Estimate (β). The estimated logistic coefficient (β) is based on the odds ratio (= 1.07) associated with 10 $\mu g/m^3$ change in TSP (Schwartz, 1993, p. 9). Assuming that PM_{10} is 55 percent of TSP[81] and that particulates greater than ten micrometers are harmless, the coefficient is calculated as follows:

$$b_{PM_{10}} = \frac{ln(1.07)}{0.55\cdot 10} = 0.0123.$$

Standard Error ($_{\beta}$) The standard error for the coefficient ($_{\beta}$) is calculated from the reported lower and upper bounds of the odds ratio (1.02 to 1.12) (Schwartz, 1993, p. 9):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.12)}{0.55\cdot 10} - \frac{\ln(1.07)}{0.55\cdot 10}\right)}{1.96} = 0.00424$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.07)}{0.55\cdot 10} - \frac{\ln(1.02)}{0.55\cdot 10}\right)}{1.96} = 0.00444$$

[81]The conversion of TSP to PM_{10} is from ESEERCO (1994, p. V-5), who cited studies by EPA (1986) and the California Air Resources Board (1982).

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.00434.$$

Population. The study population in Schwartz (1993) includes 3,874 individuals over the age of 30, living in 57 urban areas in the United States. To what extent the study should be applied to individuals under the age of 30 is unclear, and no effect is assumed for these individuals.

3.2.2 Chronic Bronchitis (Abbey et al., 1993, California)

Abbey et al. (1993) surveyed 3,914 adult Seventh Day Adventists living in California, and estimated the relationship between annual mean ambient TSP, ozone and SO_2 and the onset of certain chronic respiratory symptoms (including airway obstructive disease (AOD), chronic bronchitis, and asthma) that were not present at the beginning of the study. The initial survey was conducted in 1977 and the final survey in 1987. To ensure a better estimate of exposure, the study participants had to have been living in the same area for an extended period of time. TSP was significantly linked to new cases of AOD and chronic bronchitis, but not to asthma or the severity of asthma. Ozone was not linked to the incidence of new cases of any endpoint, but ozone was linked to the severity of asthma. No effect was found for SO_2.

The C-R function to estimate the change in chronic bronchitis is:

$$\Delta Chronic\ Bronchitis = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = annual bronchitis incidence rate per person (Abbey et al., 1993, Table 3) = 0.00378
β = estimated PM_{10} logistic regression coefficient = 0.00932
ΔPM_{10} = change in annual average PM_{10} concentration
pop = population of ages 27 and older without chronic bronchitis[82] = 0.9465*population 27+
$_{\beta}$ = standard error of β = 0.00475

Incidence Rate. The annual incidence rate is derived by taking the number of new cases (234), dividing by the number of individuals in the sample (3,310), as reported by Abbey et al.(1993, Table 3), dividing by the ten years covered in the sample, and then multiplying by one minus the reversal rate (estimated to be 46.6% based on Abbey et al. (1995a, Table 1)). Using the same data base, Abbey et al. (1995a, Table 1) reported the incidences by three age groups (25-54, 55-74, and 75+) for "cough type" and "sputum type" bronchitis, but they did not report an overall incidence rate for bronchitis.

Coefficient Estimate (β). The estimated coefficient (β) is based on the relative risk (= 1.36) associated with 60 $\mu g/m^3$ change in TSP (Abbey et al., 1993, Table 5). Assuming that PM_{10} is 55 percent of TSP[83] and that particulates greater than ten micrometers are harmless, the coefficient is calculated as follows:

$$b = \frac{\ln(1.36)}{0.55 \cdot 60} = 0.00932.$$

Standard Error ($_{\beta}$). The standard error for the coefficient ($_{\beta}$) is calculated from the reported significance level of $p < 0.05$ (Abbey et al., 1993, Table 5). We assume that $p = 0.05$, which implies that the standard error is roughly one half of the coefficient value:

$$s_b = \frac{b}{1.96} = \frac{0.00932}{1.96} = 0.00475$$

[82]Using the same data set, Abbey et al. (1995a, p. 140) reported that the respondents in 1977 ranged in age from 27 to 95. Chronic bronchitis prevalence from Adams and Marano (1995, Tables 62 and 78).

[83]The conversion of TSP to PM_{10} is from ESEERCO (1994, p. V-5), who cited studies by EPA (1986) and the California Air Resources Board (1982).

3.2.3 Chronic Bronchitis (Abbey et al., 1995b, California)

Abbey et al. (1995b) examined the relationship between estimated $PM_{2.5}$ (annual mean from 1966 to 1977), PM_{10} (annual mean from 1973 to 1977) and TSP (annual mean from 1973 to 1977) and the same chronic respiratory symptoms in a sample population of 1,868 Californian Seventh Day Adventists. The initial survey was conducted in 1977 and the final survey in 1987. To ensure a better estimate of exposure, the study participants had to have been living in the same area for an extended period of time. In single-pollutant models, there was a statistically significant $PM_{2.5}$ relationship with development of chronic bronchitis, but not for AOD or asthma; PM_{10} was significantly associated with chronic bronchitis and AOD; and TSP was significantly associated with all cases of all three chronic symptoms. Other pollutants were not examined.

The C-R function to estimate the change in chronic bronchitis is:

$$\Delta Chronic\ Bronchitis = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:
y_0 = annual bronchitis incidence rate per person (Abbey et al., 1993, Table 3) = 0.00378
β = estimated $PM_{2.5}$ logistic regression coefficient = 0.0132
$\Delta PM_{2.5}$ = change in annual average $PM_{2.5}$ concentration
pop = population of ages 27 and older without chronic bronchitis[84] = 0.9465*population 27+
$_{\beta}$ = standard error of β = 0.00680

Incidence Rate. The annual incidence rate is derived by taking the number of new cases (234), dividing by the number of individuals in the sample (3,310), as reported by Abbey et al.(1993, Table 3), dividing by the ten years covered in the sample, and then multiplying by one minus the reversal rate (estimated to be 46.6% based on Abbey et al. (1995a, Table 1)). Using the same data base, Abbey et al. (1995a, Table 1) reported the incidences by three age groups (25-54, 55-74, and 75+) for "cough type" and "sputum type" bronchitis, but they did not report an overall incidence rate for bronchitis.

Coefficient Estimate (β). The estimated coefficient (β) is based on the relative risk (= 1.81) associated with 45 $\mu g/m^3$ change in $PM_{2.5}$ (Abbey et al., 1995b, Table 2). The coefficient is calculated as follows:

$$b = \frac{\ln(1.81)}{45} = 0.0132.$$

Standard Error ($_{\beta}$). The standard error for the coefficient ($_{\beta}$) is calculated from the reported lower and upper bounds of the relative risk (0.98 to 3.25) (Abbey et al., 1995b, Table 2):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(3.25)}{45} - \frac{\ln(1.81)}{45}\right)}{1.96} = 0.00664$$

[84]Using the same data set, Abbey et al. (1995a, p. 140) reported that the respondents in 1977 ranged in age from 27 to 95. Chronic bronchitis prevalence from Adams and Marano (1995, Tables 62 and 78).

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.81)}{45} - \frac{\ln(0.98)}{45}\right)}{1.96} = 0.00696$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00680.$$

3.3 HOSPITAL ADMISSIONS

There is a wealth of epidemiological information on the relationship between air pollution and hospital admissions for various respiratory and cardiovascular diseases; in addition, some studies have examined the relationship between air pollution and emergency room (ER) visits. Because most emergency room visits do not result in an admission to the hospital -- the majority of people going to the ER are treated and return home -- we treat hospital admissions and ER visits separately, taking account of the fraction of ER visits that do get admitted to the hospital, as discussed below.

Hospital admissions require the patient to be examined by a physician, and on average may represent more serious incidents than ER visits (Lipfert, 1993, p. 230). The two main groups of hospital admissions estimated in this analysis are respiratory admissions and cardiovascular admissions. There is not much evidence linking air pollution with other types of hospital admissions. The only types of ER visits that have been linked to air pollution in the U.S. or Canada are asthma-related visits.

3.3.1 Hospital Admissions for Asthma (Burnett et al., 1999, Toronto)

Burnett et al. (1999) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada from 1980 to 1994. They estimated multiple pollutant models, where pollutants for the best fitting model were chosen using stepwise regression based on AIC criterion. Asthma admissions were linked to O_3, CO, and $PM_{2.5\text{-}10}$. This C-R function is based on the results based on this three-pollutant model.

The C-R function to estimate the change in hospital admissions for asthma associated with daily changes in $PM_{10\text{-}2.5}$ is:

$$\Delta Asthma\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10-2.5}} - 1)\right] \cdot pop,$$

where:

y_0	= daily hospital admission rate for asthma per person = 4.75 E-6
β	= $PM_{10\text{-}2.5}$ coefficient = 0.00321
$\Delta PM_{10\text{-}2.5}$	= change in daily average $PM_{10\text{-}2.5}$ concentration
pop	= population of all ages
$_\beta$	= standard error of β = 0.00106

Incidence Rate. Hospital admissions for asthma (ICD-9 code: 493) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.451 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 4.00 percent increase in admissions due to a $PM_{10-2.5}$ change of 12.2 μg/m^3 (Burnett et al., 1999, Tables 1 and 5). This translates to a relative risk of 1.04. The coefficient is calculated as follows:

$$b = \frac{\ln(1.04)}{12.2} = 0.00321.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated using the t-value (t=3.04) (Burnett, 1999):

$$s_b = \frac{.00321}{3.04} = 0.00106.$$

3.3.2 Hospital Admissions for Obstructive Lung Disease (Burnett et al., 1999, Toronto)

Burnett et al. (1999) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada from1980 to 1994. They estimated multiple pollutant models, where pollutants for best fitting model were chosen using stepwise regression based on AIC criterion. Chronic obstructive pulmonary disease (COPD) was linked to O_3 and $PM_{2.5-10}$. This C-R function is based on the results of this two-pollutant model.

The C-R function to estimate the change in hospital admissions for obstructive lung disease associated with daily changes in $PM_{10-2.5}$ is:

$$\Delta Obstructive\ Lung\ Disease\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10-2.5}} - 1)\right] \cdot pop,$$

where:

y_0	= daily hospital admission rate for obstructive lung disease per person = 5.76 E-6
β	= $PM_{10-2.5}$ coefficient = 0.00310
Δ $PM_{10-2.5}$	= change in daily average $PM_{10-2.5}$ concentration
pop	= population of all ages
$_\beta$	= standard error of β = 0.00163

Incidence Rate. Hospital admissions for obstructive lung disease (ICD-9 codes: 490-492, 496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.547 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 3.86 percent increase in admissions due to a $PM_{10-2.5}$ change of 12.2 $\mu g/m^3$ (Burnett et al., 1999, Tables 1 and 5). This translates to a relative risk of 1.0386. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0386)}{12.2} = 0.00310.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated using the t-value (t=1.90) (Burnett, 1999):

$$s_b = \frac{.00310}{1.90} = 0.00163.$$

3.3.3 Hospital Admissions for Respiratory Infection (Burnett et al., 1999, Toronto)

Burnett et al. (1999) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada from1980 to 1994. They estimated multiple pollutant models, where pollutants for best fitting model were chosen using stepwise regression based on AIC criterion. Respiratory infection admissions were linked to O_3, NO_2, and $PM_{2.5}$. This C-R function is based on the results of this three-pollutant model.

The C-R function to estimate the change in hospital admissions for respiratory infection associated with daily changes in $PM_{2.5}$ is:

$$\Delta \text{ Respiratory Infection Admissions} = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for respiratory infection per person = 1.56 E-5
β = $PM_{2.5}$ coefficient = 0.00328
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration
pop = population of all ages
$_\beta$ = standard error of β = 0.000735

Incidence Rate. Hospital admissions for respiratory infection (ICD-9 codes: 464, 466, 480-487, 494) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (1.485 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 6.08 percent increase in admissions due to a $PM_{2.5}$ change of 18 $\mu g/m^3$ (Burnett, 1999, Tables 1 and 5). This translates to a relative risk of 1.0608. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0608)}{18} = 0.00328.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated using the t-value (t=4.46) (Burnett, 1999):

$$s_b = \frac{.00328}{4.46} = 0.000735.$$

3.3.4 Hospital Admissions for All Respiratory (Burnett et al., 1997, Toronto)

Burnett et al. (1997) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada during the summers of 1992-1994. All respiratory admissions were linked to COH and O_3; other PM measures were less strongly linked. In two pollutant models, they found that CO, NO_2, and SO_2 were not significant, controlling for COH. They found that O_3 was still significant, controlling for COH. This analysis used the results from the four-pollutant model ($PM_{2.5-10}$, O_3, NO_2, and SO_2) to estimate all respiratory incidence.

The C-R function to estimate the change in all respiratory hospital admissions associated with daily changes in $PM_{10-2.5}$ is:

$$\Delta All\ Respiratory\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10-2.5}} - 1)\right] \cdot pop,$$

where:

y_0	= daily hospital admission rate for all respiratory causes per person = 2.58 E-5
β	= $PM_{10-2.5}$ coefficient = 0.00147
$\Delta PM_{10-2.5}$	= change in daily average $PM_{10-2.5}$ concentration
pop	= population of all ages
$_\beta$	= standard error of β = 0.00179

Incidence Rate. Hospital admissions for all respiratory (ICD-9 codes: 464-466, 480-486, 490-494, 496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (2.452 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a relative risk of 1.007 due to a $PM_{10-2.5}$ change of 4.75 $\mu g/m^3$ (Burnett et al., 1997, Tables 2 and 6). The coefficient is calculated as follows:

$$b = \frac{\ln(1.007)}{4.75} = 0.00147.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated using the t-value (t=0.82) (Burnett et al., 1997, Table 6)

$$s_b = \frac{.00147}{0.82} = 0.00179.$$

3.3.5 Hospital Admissions for All Respiratory (Thurston et al., 1994, Toronto)

Thurston et al. (1994) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada, for six weeks in July and August 1986-1988. In single-pollutant models, ozone and various measures of PM were linked to all respiratory admissions. In two-pollutant models, ozone was still significant, but measures of PM were often not significant; only H^+ was significant. However, since H^+ exposure information is not available, this analysis used the results from a two-pollutant model ($PM_{2.5}$ and O_3) to estimate all respiratory incidence.

The C-R function to estimate the change in all respiratory hospital admissions associated with daily changes in $PM_{2.5}$ is:

$$\Delta \, All\ Respiratory\ Admissions = b \cdot \Delta PM_{2.5} \cdot pop,$$

where:
β = $PM_{2.5}$ coefficient = 1.81 E-8
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$
pop = population of all ages
$_{\beta}$ = standard error of β = 1.79 E-8 .

Coefficient Estimate (β). Based on a linear model with ozone, the daily average $PM_{2.5}$ coefficient comes from an estimated coefficient of 0.0434, which estimates admissions per $\mu g/m^3$ of $PM_{2.5}$ (Thurston et al., 1994, Table 3). The population of Toronto was estimated to be 2.4 million (U.S. EPA, 1997a, Table D-7). We estimated a coefficient estimating admissions per person per $\mu g/m^3$ of $PM_{2.5}$ as follows:

$$b = \frac{0.0434}{2{,}400{,}000} = 1.81E-8.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated in a similar fashion (Thurston et al., 1994, Table 3):

$$s_b = \frac{0.0429}{2{,}400{,}000} = 1.79E-8.$$

3.3.6 Hospital Admissions for Pneumonia (Moolgavkar et al., 1997, Minneapolis)

Moolgavkar et al. (1997) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1991. In a four pollutant model examining pneumonia admissions in Minneapolis, ozone was significant, while NO_2, SO_2, and PM_{10} were not significant. This analysis used the results from the four-pollutant model to estimate pneumonia incidence.

The C-R function to estimate the change in hospital admissions for pneumonia associated with daily changes in PM_{10} is:

$$\Delta Pneumonia\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for pneumonia per person = 5.30 E-5
β = PM_{10} coefficient = 0.000498
Δ PM_{10} = change in daily average PM_{10} concentration
pop = population of ages 65 and older
$_{\beta}$ = standard error of β = 0.000505

Incidence Rate. Hospital admissions for pneumonia (ICD-9 codes: 480-487) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.642 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 1.00 percent increase in admissions due to a PM_{10} change of 20 $\mu g/m^3$ (Moolgavkar et al., 1997, Table 4 and p. 366); the model with a 130 df smoother was reported to be optimal (p. 368). This translates to a relative risk of 1.01. The coefficient is calculated as follows:

$$b = \frac{\ln(1.01)}{20} = 0.000498.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Moolgavkar et al., 1997, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.03)}{20} - \frac{\ln(1.01)}{20}\right)}{1.96} = 0.000500$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.01)}{20} - \frac{\ln(0.99)}{20}\right)}{1.96} = 0.000510$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000505.$$

3.3.7 Hospital Admissions for COPD (Moolgavkar et al., 1997, Minneapolis)

Moolgavkar et al. (1997) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1991. No significant effect found for any pollutant; the effect for ozone was marginally significant. This analysis used the results from a three-pollutant model (O_3, CO, PM_{10}) to estimate COPD incidence.

The C-R function to estimate the change in hospital admissions for COPD associated with daily changes in PM_{10} is:

$$\Delta\, COPD\, admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for COPD per person = 3.75 E-5
β = PM_{10} coefficient = 0.000877
Δ PM_{10} = change in daily average PM_{10} concentration
pop = population of ages 65 and older
$_\beta$ = standard error of β = 0.000777

Incidence Rate. Hospital admissions for COPD (ICD-9 codes: 490-496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.454 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 1.77 percent increase in admissions due to a PM_{10} change of 20 $\mu g/m^3$ (Moolgavkar et al., 1997, Table 4 and p. 366); the model with a 100 df smoother was reported to be optimal (p. 368). This translates to a relative risk of 1.0177. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0177)}{20} = 0.000877.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Moolgavkar et al., 1997, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.049)}{20} - \frac{\ln(1.0177)}{20}\right)}{1.96} = 0.000773$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.0177)}{20} - \frac{\ln(0.987)}{20}\right)}{1.96} = 0.000781$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000777 .$$

3.3.8 Hospital Admissions for Pneumonia (Schwartz, 1994c, Minneapolis)

Schwartz (1994c) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Minneapolis-St. Paul, Minnesota, from January 1986 to December 1989. In a two-pollutant model, Schwartz found PM_{10} significantly related to pneumonia; ozone was weakly linked to pneumonia. This analysis used the results of the two pollutant model (PM_{10}, O_3) to estimate pneumonia incidence.

The C-R function to estimate the change in hospital admissions for pneumonia associated with daily changes in PM_{10} is:

$$\Delta Pneumonia\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for pneumonia per person = 5.18 E-5
β = PM_{10} coefficient = 0.00157
Δ PM_{10} = change in daily average PM_{10} concentration
pop = population of ages 65 and older
$_{\beta}$ = standard error of β = 0.000677

Incidence Rate. Hospital admissions for pneumonia (ICD-9 codes: 480-486) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.627 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with ozone, the coefficient (β) is estimated from the relative risk (1.17) associated with a 100 $\mu g/m^3$ change in exposure (Schwartz, 1994c, Table 4 and p. 369):

$$b = \frac{\ln(1.17)}{100} = 0.00157.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1994c, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.33)}{100} - \frac{\ln(1.17)}{100}\right)}{1.96} = 0.000654$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.17)}{100} - \frac{\ln(1.02)}{100}\right)}{1.96} = 0.000700$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000677.$$

3.3.9 Hospital Admissions for COPD (Schwartz, 1994c, Minneapolis)

Schwartz (1994a) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Minneapolis, MN, from January 1986 to December 1989. In single-pollutants models, Schwartz found PM_{10} significantly related to COPD, and ozone was not significantly linked to COPD. This analysis used the results of the single-pollutant model to estimate COPD incidence.

The C-R function to estimate the change in hospital admissions for COPD associated with daily changes in PM_{10} is:

$$\Delta\ COPD\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for COPD per person = 3.75 E-5
β = PM_{10} coefficient = 0.00451
$\Delta\ PM_{10}$ = change in daily average PM_{10} concentration
pop = population of ages 65 and older
$_{\beta}$ = standard error of β = 0.00138

Incidence Rate. Hospital admissions for COPD (ICD-9 codes: 490-496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.454 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a single-pollutant model, the coefficient (β) is estimated from the relative risk (1.57) associated with a 100 $\mu g/m^3$ change in exposure (Schwartz, 1994c, Table 4 and p. 369):

$$b = \frac{\ln(1.57)}{100} = 0.00451.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1994c, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(2.06)}{100} - \frac{\ln(1.57)}{100}\right)}{1.96} = 0.00139$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.57)}{100} - \frac{\ln(1.20)}{100}\right)}{1.96} = 0.00137$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00138.$$

3.3.10 Hospital Admissions for Pneumonia (Schwartz, 1994a, Birmingham)

Schwartz (1994a) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Birmingham, Alabama, from January 1986 to December 1989. In single-pollutants model, Schwartz found PM_{10} significantly related to pneumonia; ozone was not significantly linked to pneumonia. This C-R function is based on the results of the single-pollutant model to estimate pneumonia incidence.

The C-R function to estimate the change in hospital admissions for pneumonia associated with daily changes in PM_{10} is:

$$\Delta Pneumonia\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for pneumonia per person = 5.30 E-5
β = PM_{10} coefficient = 0.00174
$\Delta\ PM_{10}$ = change in daily average PM_{10} concentration
pop = population of ages 65 and older
$_{\beta}$ = standard error of β = 0.000536

Incidence Rate. Hospital admissions for pneumonia (ICD-9 codes: 480-487) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.642 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with ozone, the coefficient (β) is estimated from the relative risk (1.19) associated with a 100 $\mu g/m^3$ change in exposure (Schwartz, 1994a, Table 4):

$$b = \frac{\ln(1.19)}{100} = 0.00174.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1994a, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.32)}{100} - \frac{\ln(1.19)}{100}\right)}{1.96} = 0.000529$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.19)}{100} - \frac{\ln(1.07)}{100}\right)}{1.96} = 0.000542$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000536.$$

3.3.11 Hospital Admissions for COPD (Schwartz, 1994a, Birmingham)

Schwartz (1994a) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Birmingham, Alabama, from January 1986 to December 1989. In single-pollutants model, Schwartz found PM_{10} significantly related to COPD; ozone was not significantly linked to COPD. This C-R function is based on the results of the single-pollutant model to estimate COPD incidence.

The C-R function to estimate the change in hospital admissions for COPD associated with daily changes in PM_{10} is:

$$\Delta\, COPD\, Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for COPD per person = 3.75 E-5
β = PM_{10} coefficient = 0.00239
Δ PM_{10} = change in daily average PM_{10} concentration
pop = population of ages 65 and older
$_\beta$ = standard error of β = 0.000838

Incidence Rate. Hospital admissions for COPD (ICD-9 codes: 490-496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.454 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with ozone, the coefficient (β) is estimated from the relative risk (1.27) associated with a 100 $\mu g/m^3$ change in exposure (Schwartz, 1994a, Table 5):

$$b = \frac{\ln(1.27)}{100} = 0.00239.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1994a, Table 5):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.50)}{100} - \frac{\ln(1.27)}{100}\right)}{1.96} = 0.000849$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.27)}{100} - \frac{\ln(1.08)}{100}\right)}{1.96} = 0.000827$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000838.$$

3.3.12 Hospital Admissions for Pneumonia (Schwartz, 1994b, Detroit)

Schwartz (1994b) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Detroit, Michigan, from January 1986 to December 1989. In a two-pollutant model, Schwartz found both PM_{10} and ozone significantly linked to pneumonia and COPD; no significant link to asthma admissions was found for either pollutant. We use the results of this two-pollutant model.

The C-R function to estimate the change in hospital admissions for pneumonia associated with daily changes in PM_{10} is:

$$\Delta Pneumonia\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for pneumonia per person = 5.18 E-5
β = PM_{10} coefficient (Schwartz, 1994b, Table 4) = 0.00115
Δ PM_{10} = change in daily average PM_{10} concentration
pop = population of ages 65 and older
$_{\beta}$ = standard error of β (Schwartz, 1994b, Table 4) = 0.00039

Incidence Rate. Hospital admissions for pneumonia (ICD-9 codes: 480-486) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.627 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

3.3.13 Hospital Admissions for COPD (Schwartz, 1994b, Detroit)

Schwartz (1994b) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Detroit, Michigan, from January 1986 to December 1989. In a two-pollutant model, Schwartz found both PM_{10} and ozone significantly linked to pneumonia and COPD; no significant link to asthma admissions was found for either pollutant. We use the results of this two-pollutant model.

The C-R function to estimate the change in hospital admissions for COPD associated with daily changes in PM_{10} is:

$$\Delta\, COPD admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for COPD per person = 3.05 E-5
β = PM_{10} coefficient (Schwartz, 1994b, Table 4) = 0.00202
Δ PM_{10} = change in daily average PM_{10} concentration
pop = population of ages 65 and older
$_{\beta}$ = standard error of β (Schwartz, 1994b, Table 4) = 0.00059

Incidence Rate. Hospital admissions for COPD (ICD-9 codes: 491-492, 494-496) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.369 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

3.3.14 Hospital Admissions for All Respiratory (Schwartz, 1996, Spokane)

Schwartz (1996) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Spokane, Washington, from January 1988 to December 1990. In single pollutant models, Schwartz found that both PM_{10} and ozone were significant. In single pollutant models, Schwartz found PM_{10} was marginally significantly linked to pneumonia and ozone was not significant; no link was found to COPD for either pollutant. Two-pollutant models were not estimated because of limited overlap between PM_{10} and ozone data.

The C-R function to estimate the change in all respiratory hospital admissions associated with daily changes in PM_{10} is:

$$\Delta\ All\ Respiratory\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for all respiratory per person 65 and older = 1.187 E-4
β = PM_{10} coefficient = 0.00163
Δ PM_{10} = change in daily average PM_{10} concentration
pop = population of ages 65 and older
$_{\beta}$ = standard error of β = 0.000470

Incidence Rate. All respiratory hospital admissions (ICD-9 codes: 460-519) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (1.437 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with ozone, the coefficient (β) is estimated from the relative risk (1.085) associated with a 50 $\mu g/m^3$ change in exposure (Schwartz, 1996, Table 3):

$$b = \frac{\ln(1.085)}{50} = 0.00163.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1996, Table 3):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.136)}{50} - \frac{\ln(1.085)}{50}\right)}{1.96} = 0.000469$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.085)}{50} - \frac{\ln(1.036)}{50}\right)}{1.96} = 0.000472$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000470.$$

3.3.15 Hospital Admissions for All Respiratory (Schwartz, 1995, New Haven)

Schwartz (1996) examined the relationship between air pollution and hospital admissions for individuals 65 and older in New Haven, Connecticut, from January 1988 to December 1990. In single-pollutant models, PM_{10} and SO_2 were significant, while ozone was marginally significant. In two-pollutant models, ozone was significant in one of two models, and had stable coefficient estimates; PM_{10} was significant in two of two models, but had less stable estimates. SO_2 was significant in one of four models. The C-R function in this analysis is based on a two-pollutant model with ozone and PM_{10}.

The C-R function to estimate the change in all respiratory hospital admissions associated with daily changes in PM_{10} is:

$$\Delta \, All\ Respiratory\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admissions for all respiratory per person 65 and older = 1.187 E-4
β = PM_{10} coefficient = 0.00172
Δ PM_{10} = change in daily average PM_{10} concentration
pop = population of ages 65 and older
$_{\beta}$ = standard error of β = 0.000930

Incidence Rate. All respiratory hospital admissions (ICD-9 codes: 460-519) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the national annual number of first-listed diagnoses for discharges (1.437 million) divided by the 1994 U.S. population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with ozone, the daily average coefficient (β) is estimated from the relative risk (1.09) associated with a change in PM_{10} exposure of 50 $\mu g/m^3$ (Schwartz, 1995, Table 3):

$$b = \frac{\ln(1.09)}{50} = 0.00172.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1995, Table 3).

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.20)}{50} - \frac{\ln(1.09)}{50}\right)}{1.96} = 0.000981$$

$$s_{b,low} = \frac{b - b_{low} -}{1.96} = \frac{\left(\frac{\ln(1.09)}{50} - \frac{\ln(1.00)}{50}\right)}{1.96} = 0.000879$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000930.$$

3.3.16 Hospital Admissions for All Respiratory (Schwartz, 1995, Tacoma)

Schwartz (1996) examined the relationship between air pollution and hospital admissions for individuals 65 and older in Tacoma, Washington, from January 1988 to December 1990. In single-pollutant models, PM_{10}, ozone, and SO_2 were all significant. In two-pollutant models, ozone was significant in two of two models, and had stable coefficient estimates; PM_{10} was significant in one of two models, but had less stable estimates; SO_2 was not significant in either of the two-pollutant models. The C-R function in this analysis is based on a two-pollutant model with ozone and PM_{10}.

The C-R function to estimate the change in all respiratory hospital admissions associated with daily changes in PM_{10} is:

$$\Delta \, All\ Respiratory\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admissions for all respiratory conditions per person 65 and older = 1.187 E-4
β = PM_{10} coefficient = 0.00227
Δ PM_{10} = change in daily average PM_{10} concentration
pop = population of ages 65 and older
$_{\beta}$ = standard error of β = 0.00145

Incidence Rate. All respiratory hospital admissions (ICD-9 codes: 460-519) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the national annual number of first-listed diagnoses for discharges (1.437 million) divided by the 1994 U.S. population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with ozone, the daily average coefficient (β) is estimated from the relative risk (1.12) associated with a change in PM_{10} exposure of 50 $\mu g/m^3$ (Schwartz, 1995, Table 6):

$$b = \frac{\ln(1.12)}{50} = 0.00227.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1995, Table 6):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.29)}{50} - \frac{\ln(1.12)}{50}\right)}{1.96} = 0.00144$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.12)}{50} - \frac{\ln(0.97)}{50}\right)}{1.96} = 0.00147$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.00145.$$

3.3.17 Hospital Admissions for Asthma (Sheppard et al., 1999, Seattle)

Sheppard et al. (1999) studied the relation between air pollution in Seattle and nonelderly hospital admissions for asthma from 1987 to 1994. They used air quality data for PM_{10}, $PM_{2.5}$, coarse $PM_{2.5\text{-}10}$, SO_2, ozone, and CO in a Poisson regression model with control for time trends, seasonal variations, and temperature-related weather effects. They found asthma hospital admissions associated with PM_{10}, $PM_{2.5}$, coarse $PM_{2.5\text{-}10}$, CO, and ozone. They did not observe an association for SO_2. They found PM and CO to be jointly associated with asthma admissions. The best fitting model was found using ozone. However, ozone data was only available April through October, so they did not consider ozone further. The C-R function in this analysis is based on a two-pollutant model with CO and $PM_{2.5}$.

The C-R function to estimate the change in hospital admissions for asthma associated with daily changes in $PM_{2.5}$ is:

$$\Delta Asthma\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for asthma per person = 4.52 E-6
β = $PM_{2.5}$ coefficient = 0.00227
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration
pop = population of ages less than 65
$_{\beta}$ = standard error of β = 0.000948

Incidence Rate. Hospital admissions for asthma (ICD-9 code: 493) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.375 million) divided by the 1994 population (227.210 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with CO, the daily average coefficient (β) is estimated from the relative risk (1.03) associated with a change in $PM_{2.5}$ exposure over the interquartile range of 8 to 21 $\mu g/m^3$ (Sheppard et al., 1999, Table 3 and p. 28):

$$b = \frac{\ln(1.03)}{13} = 0.00227.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Sheppard et al., 1999, p. 28):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.06)}{13} - \frac{\ln(1.03)}{13}\right)}{1.96} = 0.00113$$

$$s_{b,low} = \frac{b - b_{low} -}{1.96} = \frac{\left(\frac{\ln(1.03)}{13} - \frac{\ln(1.01)}{13}\right)}{1.96} = 0.000770$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000948.$$

3.3.18 Hospital Admissions for Cardiovascular (Schwartz, 1999, Eight Counties)

Schwartz (1999) examined the link between air pollution and cardiovascular admissions for persons 65 and older in eight U.S. counties from 1988 to 1990. They limited the analysis to CO and PM_{10}, and found that in two-pollutant models both pollutants were significant. The C-R function in this analysis is based on a two-pollutant model with CO and PM_{10}.

The C-R function to estimate the change in cardiovascular hospital admissions associated with daily changes in PM_{10} is:

$$\Delta Cardiovascular\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for cardiovascular disease per person 65 and older = 2.23 E-4
β = PM_{10} coefficient = 0.000737
ΔPM_{10} = change in daily average PM_{10} concentration
pop = population of ages 65 and older
$_{\beta}$ = standard error of β = 0.000170

Incidence Rate. Congestive heart failure hospital admissions (ICD-9 codes: 390-429) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (2.695 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves et al., 1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). In a two pollutant model with CO, the estimated coefficient (β) is based on a 1.86 percent increase in admissions due to a PM_{10} change of 25 $\mu g/m^3$ (Schwartz, 1999, p. 20).[85] This translates to a relative risk of 1.0186. The coefficient is calculated as follows:

$$b = \frac{ln(1.0186)}{25} = 0.000737.$$

[85]This result is based on the five counties with a PM_{10}-CO correlation of less than 0.5.

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1999, p. 20):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.0271)}{25} - \frac{\ln(1.0186)}{25}\right)}{1.96} = 0.000170$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.0186)}{25} - \frac{\ln(1.0101)}{25}\right)}{1.96} = 0.000171$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000170.$$

3.3.19 Hospital Admissions for Cardiovascular (Schwartz, 1997, Tucson)

Schwartz (1997) examined the relation between air pollution and cardiovascular admissions for persons 65 and older in Tucson, Arizona from 1988 to 1990. They focused on ozone, CO, SO_2, NO_2, and PM_{10}. In a model with the two pollutants, CO and PM_{10} were both significant. No effect was seen for O_3, SO_2, and NO_2. The C-R function in this analysis is based on a two-pollutant model with CO and PM_{10}.

The C-R function to estimate the change in daily cardiovascular hospital admissions associated with daily changes in PM_{10} is:

$$\Delta Cardiovascular\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for cardiovascular disease per person 65 and older = 2.23 E-4
β = PM_{10} coefficient = 0.00102
ΔPM_{10} = change in daily average PM_{10} concentration
pop = population of ages 65 and older
$_{\beta}$ = standard error of β = 0.000423

Incidence Rate. Congestive heart failure hospital admissions (ICD-9 codes: 390-429) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (2.695 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves et al., 1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). In a two pollutant model with CO, the estimated coefficient (β) is based on a 2.37 percent increase in admissions due to an interquartile PM_{10} change of 28 to 51 $\mu g/m^3$ (Schwartz, 1997, Tables 1 and 4). This translates to a relative risk of 1.0237. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0237)}{23} = 0.00102.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz, 1997, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.0472)}{23} - \frac{\ln(1.0237)}{23}\right)}{1.96} = 0.000503$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.0237)}{23} - \frac{\ln(1.0008)}{23}\right)}{1.96} = 0.000343$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000423.$$

3.3.20 Hospital Admissions for Cardiac (Burnett et al., 1997, Toronto)

Burnett et al. (1997) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada during the summers of1992-1994. COH and ozone were significantly linked to cardiac admissions; other PM measures less strongly linked. In two-pollutant models, they found CO, NO_2, and SO_2 were not significant, when controlling for COH. Ozone was significant, controlling for COH. In four-pollutant models, COH and O_3 were both significant; no effect for NO_2 and SO_2. The C-R function in this analysis is based on a two-pollutant model with ozone and $PM_{2.5-10}$.

The C-R function to estimate the change in cardiac hospital admissions associated with daily changes in $PM_{10-2.5}$ is:

$$\Delta \, Cardiac \; Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10-2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for cardiac problems per person = 3.81 E-5
β = $PM_{10-2.5}$ coefficient = 0.00704
$\Delta PM_{10-2.5}$ = change in daily average $PM_{10-2.5}$ concentration
pop = population of all ages
$_\beta$ = standard error of β = 0.00215

Incidence Rate. Hospital admissions for cardiac (410-414, 427-428) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (3.617 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a relative risk of 1.034 due to a $PM_{10-2.5}$ change of 4.75 $\mu g/m^3$ (Burnett et al., 1997, Tables 2 and 5). The coefficient is calculated as follows:

$$b = \frac{\ln(1.034)}{4.75} = 0.00704.$$

Standard Error ($_\beta$). The standard error ($_\beta$) was calculated using the t-value (t=3.28) (Burnett et al., 1997, Table 6)

$$s_b = \frac{.00704}{3.28} = 0.00215.$$

3.3.21 Hospital Admissions for Ischemic Heart Disease (Schwartz et al., 1995)

Schwartz and Morris (1995) examined the relationship between air pollution and hospital admissions for ischemic heart disease, dysrhythmias, and congestive heart failure in Detroit, Michigan, from 1986 to 1989. In their analysis, they considered ozone, CO, SO_2, and PM_{10}. For ischemic heart disease, they found no effect for SO_2 and ozone; however, in a two-pollutant model, they found that PM_{10} and CO were both significant. They did not find any significant relation between air pollution and dysrhythmias. For congestive heart failure, they found single-pollutant models with PM_{10} and CO were both significant; SO_2 and O_3 were not significant. In two-pollutant models, they found that PM_{10} and CO were both significant. The C-R function in this analysis is based on a two-pollutant model with CO and PM_{10}.

The C-R function to estimate the change in daily hospital admissions for ischemic heart disease associated with daily changes in PM_{10} is:

$$\Delta Ischemic\ Heart\ Disease\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for ischemic heart disease per person 65 and older = 9.96 E-5
β = PM_{10} coefficient= 0.000496
ΔPM_{10} = change in daily average PM_{10} concentration
pop = population of ages 65 and older
$_{\beta}$ = standard error of β = 0.000220

Incidence Rate. Ischemic heart disease hospital admissions (ICD-9 codes: 410-414) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (1.206 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves et al., 1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with CO, the daily average coefficient (β) is estimated from the relative risk (1.016) associated with a change in PM_{10} exposure over the interquartile range of 30 to 62 $\mu g/m^3$ (Schwartz et al., 1995, Tables 1 and 4):

$$b = \frac{\ln(1.016)}{32} = 0.000496.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz et al., 1995, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.030)}{32} - \frac{\ln(1.016)}{32}\right)}{1.96} = 0.000218$$

$$s_{b,low} = \frac{b - b_{low} -}{1.96} = \frac{\left(\frac{\ln(1.016)}{32} - \frac{\ln(1.002)}{32}\right)}{1.96} = 0.000221$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000220.$$

3.3.22 Hospital Admissions for Congestive Heart Failure (Schwartz et al., 1995)

Schwartz and Morris (1995) examined the relationship between air pollution and hospital admissions for ischemic heart disease, dysrhythmias, and congestive heart failure in Detroit, Michigan, from 1986 to 1989. In their analysis, they considered ozone, CO, SO_2, and PM_{10}. For ischemic heart disease, they found no effect for SO_2 and ozone; however, in a two-pollutant model, they found that PM_{10} and CO were both significant. They did not find any significant relation between air pollution and dysrhythmias. For congestive heart failure, they found single-pollutant models withPM_{10} and CO were both significant; SO_2 and O_3 were not significant. In two-pollutant models, they found that PM_{10} and CO were both significant. The C-R function in this analysis is based on a two-pollutant model with CO and PM_{10}.

The C-R function to estimate the change in daily hospital admissions for congestive heart failure associated with daily changes in PM_{10} is:

$$\Delta Congestive\,Heart\,Failure\,Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for congestive heart failure per person 65 and older = 5.82 E-5
β = PM_{10} coefficient = 0.000741
ΔPM_{10} = change in daily average PM_{10} concentration
pop = population of ages 65 and older
$_{\beta}$ = standard error of β = 0.000311

Incidence Rate. Congestive heart failure hospital admissions (ICD-9 code: 428) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.705 million) divided by the 1994 population of individuals 65 years and older (33.162 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves et al., 1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). Based on a model with CO, the daily average coefficient (β) is estimated from the relative risk (1.024) associated with a change in PM_{10} exposure over the interquartile range of 30 to 62 $\mu g/m^3$ (Schwartz et al., 1995, Tables 1 and 6):

$$b = \frac{\ln(1.024)}{32} = 0.000741.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated as the average of the standard errors implied by the reported lower and upper bounds of the relative risk (Schwartz et al., 1995, Table 6):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.044)}{32} - \frac{\ln(1.024)}{32}\right)}{1.96} = 0.000308$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.024)}{32} - \frac{\ln(1.004)}{32}\right)}{1.96} = 0.000314$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.000311.$$

3.3.23 Hospital Admissions for Dysrhythmias (Burnett et al., 1999, Toronto)

Burnett et al. (1999) examined the relationship between air pollution and hospital admissions for individuals of all ages in Toronto, Canada from1980 to 1994. They estimated multiple pollutant models, where pollutants for best fitting model were chosen using stepwise regression based on AIC criterion. Dysrhythmias admissions were linked to O_3, CO, and $PM_{2.5}$. This C-R function is based on the results of this three-pollutant model.

The C-R function to estimate the change in hospital admissions for dysrhythmias associated with daily changes in $PM_{2.5}$ is:

$$\Delta Dysrhythmias\ Admissions = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = daily hospital admission rate for dysrhythmias per person = 6.46 E-6
β = $PM_{2.5}$ coefficient = 0.00136
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration
pop = population of all ages
$_{\beta}$ = standard error of β = 0.000910

Incidence Rate. Hospital admissions for dysrhythmias (ICD-9 code: 427) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.614 million) divided by the 1994 population (260.372 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The estimated coefficient (β) is based on a 2.47 percent increase in admissions due to a $PM_{2.5}$ change of 18 $\mu g/m^3$ (Burnett, 1999, Tables 1 and 5). This translates to a relative risk of 1.0247. The coefficient is calculated as follows:

$$b = \frac{\ln(1.0247)}{18} = 0.00136.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) was calculated using the t-value (t=1.49) (Burnett, 1999):

$$s_b = \frac{.00136}{1.49} = 0.000910.$$

3.4 EMERGENCY ROOM VISITS

There is a wealth of epidemiological information on the relationship between air pollution and hospital admissions for various respiratory and cardiovascular diseases; in addition, some studies have examined the relationship between air pollution and ER visits. Because most ER visits do not result in an admission to the hospital -- the majority of people going to the ER are treated and return home -- we treat hospital admissions and ER visits separately, taking account of the fraction of ER visits that do get admitted to the hospital, as discussed below.

The only types of ER visit that have been explicitly linked to ozone in U.S. and Canadian epidemiological studies are asthma visits. However, it seems likely that ozone may be linked to other types of respiratory-related ER visits.

3.4.1 Emergency Room Visits for Asthma (Schwartz et al., 1993, Seattle)

Schwartz et al. (1993) examined the relationship between air quality and emergency room visits for asthma in persons under 65 and 65 and over, living in Seattle from September 1989 to September 1990. Using single-pollutant models they found daily levels of PM_{10} linked to ER visits in individuals ages under 65, and they found no effect in individuals ages 65 and over. They did not find a significant effect for SO_2 and ozone in either age group. The results of the single pollutant model for PM_{10} are used in this analysis.

The C-R function to estimate the change in daily emergency room visits for asthma associated with daily changes in PM_{10} is:

$$\Delta\ Asthma\ ER visits = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{10}} - 1)\right] \cdot pop,$$

where:

y_0 = daily ER visits for asthma per person under 65 years old = 7.69 E-6
β = PM_{10} coefficient (Schwartz et al., 1993, p. 829) = 0.00367
ΔPM_{10} = change in daily average PM_{10} concentration
pop = population of ages 0-64
$_{\beta}$ = standard error of β (Schwartz et al., 1993, p. 829) = 0.00126

Incidence Rate. Smith et al. (1997, p. 789) reported that in 1987 there were 445,000 asthma admissions and 1.2 million asthma ER visits. Assuming that all asthma hospital admissions pass through the ER room, then 37% of ER visits end up as hospital admissions. As described below, the 1994 asthma admission rate for people less than 65 is 4.522 E-6. So one might assume, ER visits = (1/0.37)*asthma admission rate = 2.7*asthma admission rate = 1.22 E-5. Now, ER visits (subtracting out those visits that end up as admissions)= 1.7*asthma admission rate = 7.69 E-6.

Asthma hospital admissions (ICD-9 code: 493) are based on first-listed discharge figures for the latest available year, 1994. The rate equals the annual number of first-listed diagnoses for discharges (0.375 million) divided by the 1994 population of individuals under 65 years old (227.21 million), and then divided by 365 days in the year. The discharge figures are from Graves and Gillum (Graves et al., 1997, Table 1), and the population data are from U.S. Bureau of the Census (1997, Table 14).

3.5 ACUTE MORBIDITY

In addition to chronic illnesses and hospital admissions, there is a considerable body of scientific research that has estimated significant relationships between elevated air pollution levels and other morbidity health effects. Chamber study research has established relationships between specific air pollution chemicals and symptoms such as coughing, pain on deep inspiration, wheezing, eye irritation and headaches. In addition, epidemiological research has found air pollution relationships with acute infectious diseases (e.g., bronchitis, sinusitis) and a variety of "symptom-day" categories. Some "symptom-day" studies examine excess incidences of days with identified symptoms such as wheezing, coughing, or other specific upper or lower respiratory symptoms. Other studies estimate relationships for days with a more general description of days with adverse health impacts, such as "respiratory restricted activity days" or work loss days.

A challenge in preparing an analysis of the minor morbidity effects is identifying a set of effect estimates that reflects the full range of identified adverse health effects but avoids double counting. From the definitions of the specific health effects examined in each research project, it is possible to identify a set of effects that are non-overlapping, and can be ultimately treated as additive in a benefits analysis.

3.5.1 Acute Bronchitis C-R Function (Dockery et al., 1996)

Dockery et al. (1996) examined the relationship between PM and other pollutants on the reported rates of asthma, persistent wheeze, chronic cough, and bronchitis, in a study of 13,369 children ages 8-12 living in 24 communities in U.S. and Canada. Health data were collected in 1988-1991, and single-pollutant models were used in the analysis to test a number of measures of particulate air pollution. Dockery et al. found that annual level of sulfates and particle acidity were significantly related to bronchitis, and $PM_{2.1}$ and PM_{10} were marginally significantly related to bronchitis.[86] They also found nitrates were linked to asthma, and sulfates linked to chronic phlegm. It is important to note that the study examined annual pollution exposures, and the authors did not rule out that acute (daily) exposures could be related to asthma attacks and other acute episodes.

[86] The original study measured $PM_{2.1}$, however when using the study's results we use $PM_{2.5}$. This makes only a negligible difference, assuming that the adverse effects of $PM_{2.1}$ and $PM_{2.5}$ are comparable.

Earlier work, by Dockery et al. (1989), based on six U.S. cities, found acute bronchitis and chronic cough significantly related to PM_{15}. Because it is based on a larger sample, the Dockery et al. (1996) study is the better study to develop a C-R function linking $PM_{2.5}$ with bronchitis. The C-R function to estimate the change in acute bronchitis is:

$$\Delta Acute\ Bronchitis = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{2.5}\cdot b} + y_0} - y_0\right]\cdot pop\ ,$$

where:

y_0 = annual bronchitis incidence rate per person = 0.044
β = estimated $PM_{2.5}$ logistic regression coefficient = 0.0272
$\Delta PM_{2.5}$ = change in annual average $PM_{2.5}$ concentration
pop = population of ages 8-12
$_{\beta}$ = standard error of β = 0.0171

Incidence Rate. Bronchitis was counted in the study only if there were "reports of symptoms in the past 12 months" (Dockery et al., 1996, p. 501). It is unclear, however, if the cases of bronchitis are acute and temporary, or if the bronchitis is a chronic condition. Dockery et al. found no relationship between PM and chronic cough and chronic phlegm, which are important indicators of chronic bronchitis. For this analysis, we assumed that the C-R function based on Dockery et al. is measuring acute bronchitis.

In 1994, 2,115,000 children ages 5-17 experienced acute conditions (Adams et al., 1995, Table 6) out of population of 48.110 million children ages 5-17 (U.S. Bureau of the Census, 1998, Table 14), or 4.4 percent of this population. This figure is somewhat lower than the 5.34 percent of children under the age of 18 reported to have chronic bronchitis in 1990-1992 (Collins, 1997, Table 8). Dockery et al. (1996, p. 503) reported that in the 24 study cities the bronchitis rate varied from three to ten percent. Finally a weighted average of the incidence rates in the six cities in the Dockery et al. (1989) study is 6.34 percent , where the sample size from each city is used to weight the respective incidence rate (Dockery et al., 1989, Tables 1 and 4).[87] This analysis assumes a 4.4 percent prevalence rate is the most representative of the national population. Note that this measure reflects the fraction of children that have a chest ailment diagnosed as bronchitis in the past year, not the number of days that children are adversely affected by acute bronchitis.[88]

Coefficient Estimate (β). The estimated logistic coefficient (β) is based on the odds ratio (= 1.50) associated with being in the most polluted city ($PM_{2.1}$ = 20.7 $\mu g/m^3$) versus the least polluted city ($PM_{2.1}$ = 5.8 $\mu g/m^3$) (Dockery et al., 1996, Tables 1 and 4). The original study used $PM_{2.1}$, however, we use the $PM_{2.1}$ coefficient and apply it to $PM_{2.5}$ data.

$$b_{PM_{2.5}} = \frac{\ln(1.50)}{(20.7-5.8)} = 0.0272\ .$$

[87]The unweighted average of the six city rates is 0.0647.

[88]In 1994, there were 13,707,000 restricted activity days associated with acute bronchitis, and 2,115,000 children (ages 5-17) experienced acute conditions (Adams et al., 1995, Tables 6 and 21). On average, then, each child with acute bronchitis suffered 6.48 days.

Standard Error ($_{\beta}$). The standard error of the coefficient ($_{\beta}$) is calculated from the reported lower and upper bounds of the odds ratio (Dockery et al., 1996, Table 4):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(2.47)}{14.9} - \frac{\ln(1.50)}{14.9}\right)}{1.96} = 0.0171$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.50)}{14.9} - \frac{\ln(0.91)}{14.9}\right)}{1.96} = 0.0171$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.0171.$$

3.5.2 Lower Respiratory Symptoms (Schwartz et al., 1994)

Schwartz et al. (1994) used logistic regression to link lower respiratory symptoms in children with SO_2, NO_2, ozone, PM_{10}, $PM_{2.5}$, sulfate and H^+ (hydrogen ion). Children were selected for the study if they were exposed to indoor sources of air pollution: gas stoves and parental smoking. The study enrolled 1,844 children into a year-long study that was conducted in different years (1984 to 1988) in six cities. The students were in grades two through five at the time of enrollment in 1984. By the completion of the final study, the cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

In single pollutant models SO_2, NO_2, $PM_{2.5}$, and PM_{10} were significantly linked to cough. In two-pollutant models, PM_{10} had the most consistent relationship with cough; ozone was marginally significant, controlling for PM_{10}. In models for upper respiratory symptoms, they reported a marginally significant association for PM_{10}. In models for lower respiratory symptoms, they reported significant single-pollutant models, using SO_2, O_3, $PM_{2.5}$, PM_{10}, SO_4, and H^+.

The C-R function used to estimate the change in lower respiratory symptoms is:

$$\Delta Lower\ Respiratory\ Symptoms = -\left[\frac{y_0}{\left(1-y_0\right)\cdot e^{\Delta PM_{2.5}\cdot b} + y_0} - y_0\right]\cdot pop\,.$$

where:

y_0 = daily lower respiratory symptom incidence rate per person = 0.0012
β = estimated $PM_{2.5}$ logistic regression coefficient = 0.01823
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration
pop = population of ages 7-14
$_\beta$ = standard error of β = 0.00586

Incidence Rate. The proposed incidence rate, 0.12 percent, is based on the percentiles in Schwartz et al. (Schwartz et al., 1994, Table 2). They did not report the mean incidence rate, but rather reported various percentiles from the incidence rate distribution. The percentiles and associated values are 10^{th} = 0 percent, 25^{th} = 0 percent, 50^{th} = 0 percent, 75^{th} = 0.29 percent, and 90^{th} = 0.34 percent. The most conservative estimate consistent with the data are to assume the incidence is zero up to the 75^{th} percentile, a constant 0.29 percent between the 75^{th} and 90^{th} percentiles, and a constant 0.34 percent between the 90^{th} and 100^{th} percentiles. Alternatively, assuming a linear slope between the 50^{th} and 75^{th}, 75^{th} and 90^{th}, and 90^{th} to 100^{th} percentiles, the estimated mean incidence rate is 0.12 percent,[89] which is used in this analysis.

Coefficient Estimate (β). The coefficient β is calculated from the reported odds ratio (= 1.44) in a single-pollutant model associated with a 20 $\mu g/m^3$ change in $PM_{2.5}$ (Schwartz et al., 1994, Table 5):

$$b = \frac{\ln(1.44)}{20} = 0.01823\,.$$

Standard Error ($_\beta$). The standard error for the coefficient ($_\beta$) is calculated from the reported lower and upper bounds of the odds ratio (Schwartz et al., 1994, Table 5):

[89]For example, the 62.5^{th} percentile would have an estimated incidence rate of 0.145 percent.

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(1.82)}{20} - \frac{\ln(1.44)}{20}\right)}{1.96} = 0.00597$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{\ln(1.44)}{20} - \frac{\ln(1.15)}{20}\right)}{1.96} = 0.00574$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.00586.$$

Population. Schwartz et al. (1994, Table 5 and p. 1235) enrolled 1,844 children into a year-long study that was conducted in different years in different cities; the students were in grades two through five and lived in six U.S. cities. All study participants were enrolled in September 1984; the actual study was conducted in Watertown, MA in 1984/85; Kingston-Harriman, TN, and St. Louis, MO in 1985/86; Steubenville, OH, and Portage, WI in 1986/87; and Topeka, KS in 1987/88. The study does not publish the age range of the children when they participated. As a result, the study is somewhat unclear about the appropriate age range for the resulting C-R function. If all the children were in second grade in 1984 (ages 7-8) then the Topeka cohort would be in fifth grade (ages 10-11) when they participated in the study. It appears from the published description, however, that the students were in grades two through five in 1984.[90] By the completion of the study, some students in the Topeka cohort would then be in the eighth grade (ages 13-14); this suggests an age range of 7 to 14.

[90]Neas et al. (1994, p. 1091) used the same data set; their description suggests that grades two to five were represented initially.

3.5.3 Upper Respiratory Symptoms (Pope et al., 1991)

Using logistic regression, Pope et al. (1991) estimated the impact of PM_{10} on the incidence of a variety of minor symptoms in 55 subjects (34 "school-based" and 21 "patient-based") living in the Utah Valley from December 1989 through March 1990. The children in the Pope et al. study were asked to record respiratory symptoms in a daily diary. With this information, the daily occurrences of upper respiratory symptoms (URS) and lower respiratory symptoms (LRS) were related to daily PM_{10} concentrations. Pope et al. describe URS as consisting of one or more of the following symptoms: runny or stuffy nose; wet cough; and burning, aching, or red eyes. Levels of ozone, NO_2, and SO_2 were reported low during this period, and were not included in the analysis. The sample in this study is relatively small and is most representative of the asthmatic population, rather than the general population. The school-based subjects (ranging in age from 9 to 11) were chosen based on "a positive response to one or more of three questions: ever wheezed without a cold, wheezed for 3 days or more out of the week for a month or longer, and/or had a doctor say the 'child has asthma' (Pope et al., 1991, p. 669)." The patient-based subjects (ranging in age from 8 to 72) were receiving treatment for asthma and were referred by local physicians. Regression results for the school-based sample (Pope et al., 1991, Table 5) show PM_{10} significantly associated with both upper and lower respiratory symptoms. The patient-based sample did not find a significant PM_{10} effect. The results from the school-based sample are used here.

The C-R function used to estimate the change in upper respiratory symptoms is:

$$\Delta Upper\ \mathrm{Re}spiratory\ Symptoms = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot b} + y_0} - y_0\right]\cdot pop\,,$$

where:

y_0 = daily upper respiratory symptom incidence rate per person = 0.3419
β = estimated PM_{10} logistic regression coefficient (Pope et al., 1991, Table 5) = 0.0036
ΔPM_{10} = change in daily average PM_{10} concentration
pop = asthmatic population[91] ages 9 to 11 = 6.91% of population ages 9 to 11
$_{\beta}$ = standard error of β (Pope et al., 1991, Table 5) = 0.0015

Incidence Rate. The incidence rate is published in Pope et al. (Pope et al., 1991, Table 2). Taking a sample-size-weighted average, one gets an incidence rate of 0.3419.

[91]Adams (1995, Table 57) reported that in 1994, 6.91% of individuals under the age of 18 have asthma.

3.5.4 Any of 19 Respiratory Symptoms (Krupnick et al., 1990)

Krupnick et al. (1990) estimated the impact of air pollution on the incidence of any of 19 respiratory symptoms or conditions in 570 adults and 756 children living in three communities in Los Angeles, California from September 1978 to March 1979. Krupnick et al. (1990) listed 13 specific "symptoms or conditions": head cold, chest cold, sinus trouble, croup, cough with phlegm, sore throat, asthma, hay fever, doctor-diagnosed ear infection, flu, pneumonia, bronchitis, and bronchiolitis. The other six symptoms or conditions are not specified.

In their analysis, they included COH, ozone, NO_2, and SO_2, and they used a logistic regression model that takes into account whether a respondent was well or not the previous day. A key difference between this and the usual logistic model, is that the model they used includes a lagged value of the dependent variable. In single-pollutant models, daily O_3, COH, and SO_2 were significantly related to respiratory symptoms in adults. Controlling for other pollutants, they found that ozone was still significant. The results were more variable for COH and SO_2, perhaps due to collinearity. NO_2 had no significant effect. No effect was seen in children for any pollutant. The results from the two-pollutant model with COH and ozone are used to develop a C-R function.

The C-R function used to estimate ARD2 is based on Krupnick et al. (1990, p. 12):[92]

$$\Delta ARD2 \cong b^{*}_{PM_{10}} \cdot \Delta PM_{10} \cdot pop\,,$$

where:

β^* = first derivative of the stationary probability = 0.000461
ΔPM_{10} = change in daily average PM_{10} concentration
pop = population of ages 18-65 (Krupnick et al., 1990, Table 1)[93]
$_{\beta}$ = standard error of β^* = 0.000239

Coefficient Estimate (β^*). The logistic regression model used by Krupnick et al. (1990) takes into account whether a respondent was well or not the previous day. Following Krupnick et al. (p. 12), the probability that one is sick is on a given day is:

$$probability(ARD2) = \frac{p_0}{1 - p_1 + p_0}$$

$$probability(ARD2 \mid sickness\ or\ not_{t-1}) = p_i = \frac{1}{1 - e^{b_0 + b_1 \cdot ARD2_{t-1} + X \cdot b}}\,,\ for\ i = 0,1.$$

[92]Krupnick and Kopp (1988, p. 2-24) and ESEERCO (1994, p. V-32) used the same C-R functional form as that used here.

[93]Krupnick et al. (1990, Table 1) reported the age distribution in their complete data, but they did not report the ages of individuals that were considered "adult." This analysis assumes that individuals 18 and older were considered adult. Only a small percentage (0.6%) of the study population is above the age of 60, so the C-R function was limited to the adult population up through the age of 65.

where:

X	=	the matrix of explanatory variables
p_0	=	the probability of sickness on day t, given wellness on day t-1, and
p_1	=	the probability of sickness on day t, given sickness on day t-1.

In other words, the transition probabilities are estimated using a logistic function; the key difference between this and the usual logistic model, is that the model includes a lagged value of the dependent variable.

To calculate the impact of COH (or other pollutants) on the probability of ARD2, it is possible, in principle, to estimate ARD2 before the change in COH and after the change:

$$\Delta ARD2 = ARD2_{after} - ARD2_{before} .$$

However the full suite of coefficient estimates are not available.[94] Rather than use the full suite of coefficient values, the impact of COH on the probability of probability of ARD2 may be approximated by the derivative of ARD2 with respect to COH:

$$\frac{\partial probability(ARD2)}{\partial COH} = \frac{p_0 \cdot (1 - p_1) \cdot b_{COH} \cdot \left[p_1 + (1 - p_0)\right]}{(1 - p_1 + p_0)^2} = b^*_{COH} ,$$

where β_{COH} is the reported logistic regression coefficient for COH. Since COH data are not available for the benefits analysis, an estimated PM_{10} logistic regression coefficient is used based on the following assumed relationship between PM_{10}, COH, and TSP:

$$COH = 0.116 \cdot TSP$$

$$PM_{10} = 0.55 \cdot TSP$$

$$\Rightarrow COH = 0.2109 \cdot PM_{10}$$

$$\Rightarrow b_{PM_{10}} = 0.2109 \cdot b_{COH} = 0.2109 \cdot 0.0088 = 0.001856 .$$

This analysis uses $\beta_{COH} = 0.0088$ (Krupnick et al., 1990, Table V equation 3). The conversion from COH to TSP is based on study-specific information provided to ESEERCO (1994, p. V-32). The

[94]The model without NO_2 (Krupnick et al., 1990, Table V equation 3) was used in this analysis, but the full suite of coefficient estimates for this model were not reported. Krupnick et al. (1990, Table IV) reported all of the estimated coefficients for a model of children and for a model of adults when four pollutants were included (ozone, COH, SO_2, and NO_2). However, because of high collinearity between NO_2 and COH, NO_2 was dropped from some of the reported analyses (Krupnick et al., p. 10), and the resulting coefficient estimates changed substantially (see Krupnick et al., 1990, Table IV). Both the ozone and COH coefficients dropped by about a factor of two or more.

conversion of TSP to PM_{10} is from also from ESEERCO (1994, p. V-5), which cited studies by EPA (1986) and the California Air Resources Board (1982).

The change in the incidence of ARD2 associated with a given change in COH is then estimated by:

$$\frac{\P ARD2}{\P PM_{10}} \cong \frac{\Delta ARD2}{\Delta PM_{10}}$$

$$\Rightarrow \frac{\Delta ARD2}{\Delta PM_{10}} \cong b^{*}_{PM_{10}}$$

$$\Rightarrow \Delta ARD2 \cong b^{*}_{PM_{10}} \cdot \Delta PM_{10} .$$

This analysis uses transition probabilities obtained from Krupnick et al. as reported by ESEERCO (1994, p. V-32), for the adult population: $p_1 = 0.7775$ and $p_0 = 0.0468$. This implies:

$$b^{*}_{PM_{10}} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.001856 \cdot \left[0.7775_1 + (1 - 0.0468)\right]}{(1 - 0.7775 + 0.0468)^2} = 0.000461.$$

Standard Error ($_{\beta}$). The standard error for the coefficient ($_{\beta}$) is derived using the reported standard error of the logistic regression coefficient in Krupnick et al. (1990, Table V):

$$\Rightarrow b_{PM_{10},\,high} = 0.2109 \cdot b_{COH,\,high} = 0.2109 \cdot \left(0.0088 + (1.96 \cdot 0.0046)\right) = 0.003757$$

$$\Rightarrow b^{*}_{PM_{10},\,high} = \frac{0.0468 \cdot (1 - 0.7775) \cdot 0.003757 \cdot \left[0.7775 + (1 - 0.0468)\right]}{(1 - 0.7775 + 0.0468)^2} = 0.000934$$

$$s_{b,\,high} = \frac{b_{PM_{10},\,high} - b_{PM_{10}}}{1.96} = \frac{(0.000934 - 0.000461)}{1.96} = 0.000236$$

$$b_{PM_{10},\,low} = 0.2109 \cdot b_{COH,\,low} = 0.2109 \cdot \left(0.0088 - (1.96 \cdot 0.0046)\right) = -4.555 \cdot 10^{-5}$$

$$\Rightarrow b^{*}_{PM_{10},\,low} = \frac{0.0468 \cdot (1 - 0.7775) \cdot (-4.555 \cdot 10^{-5}) \cdot \left[0.7775 + (1 - 0.0468)\right]}{(1 - 0.7775 + 0.0468)^2} = -1.132 \cdot 10^{-5}$$

$$\Rightarrow s_{b,\,low} = \frac{b - b_{low}}{1.96} = \frac{\left(0.000461 + 1.132 \cdot 10^{-5}\right)}{1.96} = 0.000241$$

$$s_b = \frac{s_{b,high} + s_{b,low}}{2} = 0.000239 .$$

3.5.5 Shortness of Breath (Ostro et al., 1995)

Using a logistic regression estimation, Ostro et al. (1995) estimated the impact of PM_{10}, ozone, NO_2, and SO_2 on the incidence of coughing, shortness of breath, and wheezing in 83 African-American asthmatic children ages 7-12 living in Los Angeles from August through September 1992. Regression results show both PM_{10} and ozone significantly linked to shortness of breath; the beginning of an asthma episode was also significantly linked to ozone. No effect was seen for NO_2 and SO_2. Results for single-pollutant models only were presented in the published paper.

The C-R function to estimate the change in shortness of breath days is:

$$\Delta Shortness\,of\;\; Breath = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot b} + y_0} - y_0\right]\cdot pop ,$$

where:

y_0 = daily shortness of breath incidence rate per person (Ostro et al., 1995, p. 715) = 0.056
β = estimated PM_{10} logistic regression coefficient = 0.00841
ΔPM_{10} = change in daily average PM_{10} concentration
pop = asthmatic African-American population ages 7 to 12 = 6.91% of African-American population ages 7 to 12
$_{\beta}$ = standard error of β = 0.00363

Prevalence. Adams (1995, Table 57) reported that in 1994, 6.91% of individuals under the age of 18 have asthma. It has been reported that African-Americans have a higher prevalence of asthma (e.g., see U.S. EPA, 1996b). Ostro et al. (1995, p. 711) noted that "Although prevalence is only somewhat greater among African-Americans than among whites, rates of morbidity are markedly higher." Indeed, the asthma rates for whites and African-Americans were almost identical in 1994 (1995, Table 59), so no correction is made to the estimated prevalence rate for asthma in African-Americans.

Coefficient Estimate (β). The estimated logistic coefficient (β) is based on the odds ratio of 1.60 (Ostro et al., 1995, Table 3) associated with a change in mean PM_{10} of 55.87 $\mu g/m^3$ (Ostro et al., 1995, Table 2). The coefficient is calculated as follows:

$$b = \frac{\ln(1.60)}{(55.87)} = 0.00841 .$$

Standard Error ($_{\beta}$). The standard error for the coefficient ($_{\beta}$) is calculated from the reported lower and upper bounds of the odds ratio (Ostro et al., 1995, Table 2):

$$s_{b,high} = \frac{b_{high} - b}{1.96} = \frac{\left(\frac{\ln(2.37)}{55.87} - \frac{\ln(1.60)}{55.87}\right)}{1.96} = 0.003588$$

$$s_{b,low} = \frac{b - b_{low}}{1.96} = \frac{\left(\frac{ln(1.60)}{55.87} - \frac{ln(1.07)}{55.87}\right)}{1.96} = 0.003674$$

$$s_b = \frac{s_{high} + s_{low}}{2} = 0.003631.$$

3.5.6 Moderate (or Worse) Asthma (Ostro et al., 1991)

Ostro et al. (1991) examined the effect of air pollution on asthmatics, ages 18 to 70, living in Denver, Colorado from December 1987 to February 1988. The respondents in this study were asked to record daily a subjective rating of their overall asthma status each day (0=none, 1=mild, 2=moderate, 3=severe, 4=incapacitating). Ostro et al. then examined the relationship between moderate (or worse) asthma and H^+, sulfate, SO_2, $PM_{2.5}$, estimated $PM_{2.5}$, PM_{10}, nitrate, and nitric acid. Daily levels of H^+ were linked to cough, asthma, and shortness of breath. $PM_{2.5}$ was linked to asthma. Sulfate was linked to shortness of breath. No effects seen for other pollutants. The C-R function is based on a single-pollutant linear regression model where the log of the pollutant is used.

The C-R function to estimate the change in the number of days with moderate (or worse) asthma is:

$$\Delta Days\ Moderate / Worse\ Asthma = -b \cdot \ln\left(\frac{PM_{2.5,\,after}}{PM_{2.5,\,before}}\right) \cdot pop\,,$$

where:

β = estimated $PM_{2.5}$ coefficient (Ostro et al., 1991, Table 5) = 0.0006

$PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration

pop = asthmatic population of all ages = 5.61% of the population of all ages (Adams et al., 1995 Table 57)

$_{\beta}$ = standard error of β (Ostro et al., 1991, Table 5) = 0.0003

Coefficient Estimate (β). Two $PM_{2.5}$ coefficients are presented, both equal 0.0006, however only one is significant. The coefficient based on data that does not include estimates of missing $PM_{2.5}$ values is not significant ($_{\beta}$ = 0.0053); the coefficient that includes estimates of missing $PM_{2.5}$ values (estimated using a function of sulfate and nitrate) is significant at $p < 0.5$ ($_{\beta}$ = 0.0003). The latter coefficient is used here.

Population. The C-R function is applied to asthmatics of all ages, where it is assumed that 5.61 percent of the population of all ages is asthmatic. This raises two issues: the age group for which the function should be used, and the fraction of the population that is asthmatic. The study population consists of asthmatics between the ages of 18 and 70. It seems reasonable to assume that individuals over the age of 70 are at least as susceptible as individuals in the study population. It also seems reasonable to assume that individuals under the age of 18 are also susceptible. For example, controlling for oxidant levels, Whittemore and Korn (1980) found TSP significantly related to asthma attacks in a study population comprised primarily (59 percent) of individuals less than 16 years of age.

3.5.7 Minor Restricted Activity Days (Ostro et al., 1989b)

Ostro and Rothschild (1989b) estimated the impact of $PM_{2.5}$ on the incidence of minor restricted activity days (MRADs) and respiratory-related restricted activity days (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Controlling for $PM_{2.5}$, two-week average O_3 has highly variable association with RRADs and MRADs. Controlling for O_3, two-week average $PM_{2.5}$ was significantly linked to both health endpoints in most years.

The study is based on a "convenience" sample of individuals ages 18-65. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals 65 and younger. The elderly appear more likely to die due to PM exposure than other age groups (e.g., Schwartz, 1994d, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994a; Schwartz, 1994b).

Using the results of the two-pollutant model, we developed separate coefficients for each year in the analysis, which were then combined for use in this analysis. The coefficient used in this analysis is a weighted average of the coefficients (Ostro, 1987, Table IV) using the inverse of the variance as the weight. The C-R function to estimate the change in the number of minor restricted activity days (MRAD) is:

$$\Delta MRAD = \Delta y \cdot pop = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = daily MRAD daily incidence rate per person = 0.02137
β = inverse-variance weighted $PM_{2.5}$ coeffcient = 0.00741
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration[95]
pop = adult population ages 18 to 65
$_{\beta}$ = standard error of β = 0.0007

Incidence Rate. The annual incidence rate (7.8) provided by Ostro and Rothschild (1989b, p. 243) was divided by 365 to get a daily rate of 0.02137.

Coefficient Estimate (β). The coefficient is a weighted average of the coefficients in Ostro and Rothschild (1989b, Table 4) using the inverse of the variance as the weight:

$$b = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = 0.00741.$$

[95]The study used a two-week average pollution concentration; the daily rate used here is assumed to be a reasonable approximation.

Standard Error ($_{\beta}$). The standard error of the coefficient ($_{\beta}$) is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$s_b^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}}\right) = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{g}\right) = \sum_{i=1976}^{1981} \text{var}\left(\frac{b_i}{s_{b_i}^2 \cdot g}\right).$$

This reduces down to:

$$s_b^2 = \frac{1}{g} \Rightarrow s_b = \sqrt{\frac{1}{g}} = 0.00070.$$

3.5.8 Work Loss Days (Ostro, 1987)

Ostro (1987) estimated the impact of $PM_{2.5}$ on the incidence of work-loss days (WLDs), restricted activity days (RADs), and respiratory-related RADs (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average $PM_{2.5}$ levels were significantly linked to work-loss days, RADs, and RRADs, however there was some year-to-year variability in the results. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function used here is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight.

The study is based on a "convenience" sample of individuals ages 18-65. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals 65 and younger. The elderly appear more likely to die due to PM exposure than other age groups (e.g., Schwartz, 1994d, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994a; Schwartz, 1994b). On the other hand, the number of workers over the age of 65 is relatively small; it was under 3% of the total workforce in 1996 (U.S. Bureau of the Census, 1997, Table 633).

The C-R function to estimate the change in the number of work-loss days is:

$$\Delta WLD = \Delta y \cdot pop = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1)\right] \cdot pop,$$

where:

y_0 = daily work-loss-day incidence rate per person = 0.00648
β = inverse-variance weighted $PM_{2.5}$ coefficient = 0.0046
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration[96]
pop = population of ages 18 to 65
$_{\beta}$ = standard error of β = 0.00036

Incidence Rate. The estimated 1994 annual incidence rate is the annual number (376,844,000) of WLD per person in the age 18-64 population divided by the number of people in 18-64 population (159,361,000). The 1994 daily incidence rate is calculated as the annual rate divided by 365.[97] Data are from U.S. Bureau of the Census (1997, Table 14) and Adams (1995, Table 41).

[96]The study used a two-week average pollution concentration; the daily rate used here is assumed to be a reasonable approximation.

[97]Ostro (1987) analyzed a sample aged 18 to 65. It is assumed that the age 18-64 rate is a reasonably good approximation to the rate for individuals 18-65. Data are from U.S. Bureau of the Census (1997, Table 14) and Adams (1995, Table 41).

Coefficient Estimate (β). The coefficient used in the C-R function is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight:

$$b = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = 0.0046 .$$

Standard Error (s_β). The standard error of the coefficient (s_β) is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$s_b^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{g} \right) = \sum_{i=1976}^{1981} \text{var}\left(\frac{b_i}{s_{b_i}^2 \cdot g} \right).$$

This eventually reduces down to:

$$s_b^2 = \frac{1}{g} \Rightarrow s_b = \sqrt{\frac{1}{g}} = 0.00036 .$$

3.5.9 Restricted Activity Days (Ostro, 1987)

Ostro (1987) estimated the impact of $PM_{2.5}$ on the incidence of work-loss days (WLDs), restricted activity days (RADs), and respiratory-related RADs (RRADs) in a national sample of the adult working population, ages 18 to 65, living in metropolitan areas. The annual national survey results used in this analysis were conducted in 1976-1981. Ostro reported that two-week average $PM_{2.5}$ levels were significantly linked to work-loss days, RADs, and RRADs, however there was some year-to-year variability in the results. Separate coefficients were developed for each year in the analysis (1976-1981); these coefficients were pooled. The coefficient used in the concentration-response function used here is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight.

The study is based on a "convenience" sample of individuals ages 18-65. Applying the C-R function to this age group is likely a slight underestimate, as it seems likely that elderly are at least as susceptible to PM as individuals 65 and younger. The elderly appear more likely to die due to PM exposure than other age groups (e.g., Schwartz, 1994d, p. 30) and a number of studies have found that hospital admissions for the elderly are related to PM exposures (e.g., Schwartz, 1994a; Schwartz, 1994b).

The C-R function to estimate the change in the number of restricted activity days (RAD) is:

$$\Delta RAD = \Delta y \cdot pop = -\left[y_0 \cdot (e^{-b \cdot \Delta PM_{2.5}} - 1) \right] \cdot pop,$$

where:

y_0 = daily RAD incidence rate per person = 0.0177
β = inverse-variance weighted $PM_{2.5}$ coeffcient = 0.00475
$\Delta PM_{2.5}$ = change in daily average $PM_{2.5}$ concentration[98]
pop = adult population ages 18 to 65
$_{\beta}$ = standard error of β = 0.00029

Incidence Rate. The estimated daily RAD incidence rate is the 1994 annual number of RAD for the population aged 18-64 in the nation (1,029,419,000), divided by the number of people aged 18-64 in the nation (159,361,000), and then divided by 365.[99] RAD estimates are from Adams (1995, Table 21), and the 1994 population estimate is from U.S. Bureau of the Census (1997, Table 14).

Coefficient Estimate (β). The coefficient used in the C-R function is a weighted average of the coefficients in Ostro (1987, Table III) using the inverse of the variance as the weight:

[98]The study used a two-week average pollution concentration; the daily rate used here is assumed to be a reasonable approximation.

[99]Ostro (1987) analyzed a sample aged 18 to 65. It is assumed that the age 18-64 rate is a reasonably good approximation. This may be a slight underestimate, since the 65 and over rate is significantly higher than the rest of the adult population (Adams et al., 1995, Table 16). RAD estimates are from Adams and Marano (Table 21), and the 1994 population estimate is from U.S. Bureau of the Census (1997, Table 14).

$$b = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = 0.00475 .$$

Standard Error ($_{\beta}$). The standard error of the coefficient ($_{\beta}$) is calculated as follows, assuming that the estimated year-specific coefficients are independent:

$$s_b^2 = \text{var}\left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{\sum_{i=1976}^{1981} \frac{1}{s_{b_i}^2}} \right) = \left(\frac{\sum_{i=1976}^{1981} \frac{b_i}{s_{b_i}^2}}{g} \right) = \sum_{i=1976}^{1981} \text{var}\left(\frac{b_i}{s_{b_i}^2 \cdot g} \right).$$

This eventually reduces down to:

$$s_b^2 = \frac{1}{g} \Rightarrow s_b = \sqrt{\frac{1}{g}} = 0.00029 .$$

3.5.10 Asthma Attacks: Whittemore and Korn (1980)

Whittemore and Korn (1980) examined the relationship between air pollution and asthma attacks in a survey of 443 children and adults, living in six communities in southern California during three 34-week periods in 1972-1975. The analysis focused on TSP and ozone. Respirable PM, NO_2, SO_2 were highly correlated with TSP and excluded from the analysis. In a two pollutant model, daily levels of both TSP and O_x were significantly related to reported asthma attacks.

The C-R function to estimate the change in the number of asthma attacks is:

$$\Delta asthma\ attacks = -\left[\frac{y_0}{(1-y_0)\cdot e^{\Delta PM_{10}\cdot b} + y_0} - y_0\right]\cdot pop,$$

where:

y_0 = daily incidence of asthma attacks = 0.027 (Krupnick, 1988, p. 4-6)
β = PM_{10} coefficient = 0.00144
ΔPM_{10} = change in daily PM_{10} concentration
pop = population of asthmatics of all ages = 5.61% of the population of all ages (Adams et al., 1995 Table 57).
$_{\beta}$ = standard error of β = 0.000556

Incidence Rate. The annual rate of 9.9 asthma attacks per astmatic is divided by 365 to get a daily rate. A figure of 9.9 is roughly consistent with the recent statement that "People with asthma have more than 100 million days of restricted activity" each year (National Heart, 1997, p. 1). This 100 million incidence figure coupled with the 1996 population of 265,557,000 (U.S. Bureau of the Census, 1997, Table 2) and the latest asthmatic prevalence rate of 5.61% (Adams et al., 1995, Table 57), suggest an annual asthma attach rate per asthmatic of 6.7.

Coefficient Estimate (β). Based on a model with ozone, the coefficient is based on a TSP coefficient (0.00079) (Whittemore et al., 1980, Table 5). Assuming that PM_{10} is 55 percent of TSP[100] and that particulates greater than ten micrometers are harmless, the coefficient is calculated as follows:

$$b = \frac{0.00079}{0.55} = 0.00144.$$

Standard Error ($_{\beta}$). The standard error ($_{\beta}$) is calculated from the two-tailed p-value (<0.01) reported by Whittemore and Korn (1980, Table 5), which implies a t-value of at least 2.576 (assuming a large number of degrees of freedom).

$$s_b = \frac{b}{t} = \frac{0.144}{2.576} = 0.000556.$$

[100]The conversion of TSP to PM_{10} is from ESEERCO (1994, p. V-5), who cited studies by EPA (1986) and the California Air Resources Board (1982).